A POPULATION BASED SURVEY TO ASSESS THE CURRENT SCENARIO OF LEPROSY IN ALANGANALLUR SOUTH TAMIL NADU.

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CHENNAI – TAMIL NADU

CERTIFICATE FROM THE DEAN

This is to certify that this dissertation entitled **'A POPULATION BASED SURVEY TO ASSESS THE CURRENT SCENARIO OF LEPROSY IN ALANGANALLUR SOUTH TAMILNADU'** submitted by **Dr. KAVITHA V.** to The Tamil Nadu Dr.M.G.R. Medical University, Chennai is in partial fulfilment of the requirement for the award of M.D. [DERMATO VENEREO LEPROLOGY] and is a bonafide research work carried out by her under direct supervision and guidance. This work has not previously formed the basis for the award of any degree or diploma.

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ABSTRACT:

BACK GROUND :

Leprosy is a chronic granulomatous disease caused by Mycobacterium leprae. The introduction of Multidrug therapy in the year 1983 has reduced the prevalence of leprosy in Tamil Nadu from 118/10,000 in 1983 to less than 10/10,000 in 1994.

Leprosy was declared eliminated on January 2006, since the prevalence reached the level below 1/10000 population in India. As per latest National Leprosy Eradication Programme data available only 1.35 lakh new cases were reported in India during the Year 2012-2013.But several reports from Himachal Pradesh and Maharashtra had indicated that leprosy case load may be many folds higher in India and elsewhere than is being reported under respective government health systems.

Aims and objectives:

a. To assess the incidence and prevalence of leprosy in Alanganallur block of Madurai district through door to door survey.

To compare Government data which reflects the voluntarily reported cases with this survey to see whether there is any undetected cases of leprosy and hence under reporting in Health system.

Methodology:

The survey was conducted with the help of 25 Accredited Social Health Activist (ASHA) their work was supervised by Health inspectors, Non medical supervisors and Investigator. These ASHA workers were trained to do house to house survey in their villages to identify suspected cases of leprosy, leprosy-related disability and complications and old cases using verbal questionnaire. I the Investigator, examined these suspects to filter out non leprosy cases and the leprosy cases were confirmed by a thorough clinical examination, slit skin smear and biopsy when necessary.

There is a large backlog in the detection of leprosy cases in the endemic areas. There are also problems faced by patients in accessing the state healthcare facilities. This warrants the need for active surveillance in the community to attain the actual elimination

Introduction:

Leprosy is a chronic granulomatous disease caused by Mycobacterium leprae. The introduction of Multidrug therapy in the year 1983 has reduced the prevalence of leprosy in Tamil Nadu from 118/10,000 in 1983 to less than 10/10,000 in 1994.

Public Health Department conducted cost-analysis study of the National Leprosy Eradication Programme (NLEP) and concluded that the vertical programme was no longer cost-effective. Based on this report, in July 1997 Tamil Nadu was the first state in India, to integrate NLEP with the Primary Health Care system.

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This has created doubts about actual achievements in leprosy control. Hence this study was undertaken to perform door to door survey at Alanganallur block which is identified to have higher Annual New Case Detection Rate than State and National averages for past 2 years.

Aims and objectives:

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b. To compare Government data which reflects the voluntarily reported cases with this survey to see whether there is any undetected cases of leprosy and hence under reporting in Health system.

History of leprosy

The word ‘Lepra’ indicates any disease which produces scaly skin lesions. Daniel Cornelius Danielson and Carl Wilhelm Boeck, researchers of Norway, classified leprosy into two types, Elephantiasis Graecorum Tuberosa and Elepantiasis anesthetosa. In India leprosy has been mentioned in Vedic period. Leper means most sinful person.

Indian physician Dr. Gopala Krishna did a comprehensive work and prescribed ‘galita kushtari rasa’ for treatment of leprosy.

Norwegian medical society appointed Hansen to study the cause of leprosy and he proved the infectious etiology by demonstrating the rod shaped bodies from the nodules of leprosy. Thus Armauer Hansen (1841-1912) discovered leprosy bacillus. The year 1873 is considered as the year of discovery of leprosy bacillus.[2]

Sheppard in 1960 transmitted M. leprae in to animals. Ehrlich in 1882 described acid fast property of lepra bacilli.

Jeanselme of France in 1897 identified that nose was the site of entry of infection. Member of German commission named Georg Sticker visited ‘Homeless Leper Asylum’ in Mumbai to investigate leprosy and found bacillary clusters and intracellular globi in nasal smears.

Fromm and Wittman synthesized Dapsone for treatment in the year 1908. Paul Unna demonstrated cell-free sub epidermal zone or Grenz zone in histopathology of nodular leprosy in 1909.

Epidemiological determinants

Mycobacterium leprae is the causative agent for a chronic granulomatous disease called leprosy. This disease is classified into paucibacillary(PB) and multibacillary (MB) based on the number of skin lesions and nerve involvement.[3]

Prevalence:

It is defined as

$$\frac{\text{The total no of leprosy cases under treatment as on March 31st}}{\text{Population as on March 31st}} \times 10000$$

Not a good indicator for assessing the programme. It indicates only the magnitude of the disease. It is useful for assessing multi drug therapy requirement and also reflects efficiency in patient management.

Annual New Case Detection Rate:

It is defined as

$$\frac{\text{No. of New cases detected during a specified period of time}}{\text{Population in that area during a specified period}} \times 100000$$

This indicator is useful for monitoring the programme.

Proportion of child cases

Is defined as an

$$\frac{\text{Number of child cases (< 15 years) detected in a year}}{\text{Total new cases detected in a year}} \times 100$$

High proportion indicates higher transmission of the disease.

Proportion of female cases:

It is defined as

$$\frac{\text{Number of female cases detected in a year}}{\text{Total new cases detected in a year}} \times 100$$

It gives an indication whether the women have adequate access to diagnostic services.

Deformity rate:

Percentage of patients with deformities among newly detected cases in a period of one year. This indicates delay in diagnosing

It gives an indication whether the women have adequate access to diagnostic services.

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Host Factors

Seen commonly among the age groups of 20-30 years. Childhood leprosy indicates active transmission of disease. Both sexes are affected however there is a little male predominance.[12]

Increased cases are seen in urban areas due to migration of rural population. Poor hygiene and living conditions of 8 to 10 people sharing a common room are important risk factors.

Environmental Factors

Bacilli survive for about nine days outside the body (dried nasal secretions) and 46 days in moist soil at room temperature.

Social Factors

Lack of education, ventilation and personal hygiene are risk factors for easy transmission of disease.

Transmission

The natural reservoir is Man and untreated cases of leprosy are the source of infection. However some animals like armadillos, chimpanzees, Mangabey monkeys may be infected with M.leprae but transmission from animal to human is very minimal.

Nose is the major route of entry and exit of bacilli from an infected person. Modes of transmission of infection are

1. Droplet infection
2. Contact – by skin to skin contact
3. In utero Transmission
4. Transmission through Ingestion (Breast Milk) – Mothers with lepromatous leprosy.

There is a long incubation period (average of 5 to 7 years) for leprosy. [4,50]

MicrobiologicalAspects

M.leprae is a non-motile, acid-fast, obligate intracellular bacillus found within the macrophages, Schwann cells of nerves, muscle cells, endothelial cells of blood vessels, melanocytes of skin and chondrocytes of cartilages.

Cell Wall:

Contains inner electron dense and outer electron transparent layer. Inner layer has peptidoglycan, which is attached to arabinogalactan polymer modified by addition of mycolic acids. Unlike M.tuberculosis, M.leprae peptidoglycan had glycine instead of alanine found at the amino terminal. Outer transparent layer made up of lipopolysaccharides.. There are many antigens found on M.leprae, the most important being phenolic glycolpid-1 (PGL-1)[3,5] Major lipid in the cell wall and also gives immunological specificity is PGL-1.

Cell Membrane:

It contains proteins and lipids. Major membrane protein-1 & protein-2, which is responsible for active and passive transport of substances across the cell. Phospholipid is the major lipid in the cell membrane.

Cytoplasm:

28KDa, 17KDa and Heat Shock Protein are the three major protein in the cytoplasm.

Capsule:

The two main unique lipids are (i) Pthiocerol dimycocerosate (ii) Phenolic glycolipid 1 (PGL-1). Protects the bacilli from toxic effects of host lysosomal enzymes, thereby causing immune evasion.

Biological Properties

Bacilli cannot be grown in artificial culture medium. It can be grown only in the animals like footpads of mouse and armadillo. The generation time is 11 to 13 days. Temperature required for the bacilli is less than 37^{O} C. [6.]

Pathology

Indeterminate Leprosy:

One or a few vague hypo pigmented macules with or without sensory impairment. The histopathological features reveal only perivascular and periadnexal infiltrate of lymphocytes and histiocytes in the dermis with no epidermal change.

The deep dermal nerves are often thickened and show intraneural infiltration by lymphocytes. The presence of acid-fast bacilli (AFB) inside a nerve, or an erector pili muscle confirms the diagnosis.

75% of cases heal without treatment. Rest may progress to tuberculoid, lepromatous or borderline leprosy, depending on the host immune status.

Tuberculoid leprosy (TT)

Skin lesions are well-defined, 1–3 hypo pigmented hypoaesthetic having characteristic raised margin and a healing center. Skin biopsy shows tuberculoid granuloma characterized by dermal collections of epitheloid cells with Langhans giant cells surrounded by a rim of lymphocytes, which hugs the epidermis, and also seen around the perivascular and periadnexal structures.

Dermal nerves shows lymphocytic infiltrate alone or epitheloid granuloma which erodes the nerve. Multiple swelling of the cutaneous and peripheral nerve trunks with caseous necrotic material seen called “segmental necrotizing granulomatous neuritis”.

Borderline Tuberculoid Leprosy

Multiple skin lesions with nerve involvement, which on Histopathological examination revealed large number of epitheloid granulomas with fewer giant cells and lymphocytes when compared to tuberculoid. Nerves are infiltrated with granuloma and later destruction of nerves occurs. Occasional bacilli may be seen in nerves on careful examination.[7]

Mid-Borderline (BB) Leprosy

Histopathological examination shows diffuse collections of epitheloid cells and some macrophages in the dermis which is separated from the overlying atrophic epidermis by a clear zone. Transverse section of a nerve shows “cut onion” appearance due to proliferation of perineural cells in an attempt to repair.

Borderline lepromatous leprosy.

Histopathological examination shows atrophic epidermis with grenz zone which separates the epidermis from the infiltrate composed primarily of foamy macrophages unevenly mixed with lymphocytes. Acid fast bacilli staining reveals numerous bacilli inside macrophages and Schwann cells.

Lepromatous Leprosy

Epidermis is always atrophic with underlying grenz zone. Dermal infiltrate composed entirely by foamy macrophages with minimal lymphocytes. There is perineural infiltration by macrophages. Acid fast bacilli staining revealed clumps of bacilli in Schwann cells, endothelial cells etc. [7,8]

Case definitions of leprosy:

Leprosy case is defined as an individual who has not completed the course of treatment and has one or more of the three cardinal features:

1) Hypo pigmented or erythematous skin lesions with definite loss of sensation.
2) Involvement of the peripheral nerves, by definite thickening with sensory impairment.
3) AFB positive skin smear. [3]

Classification of leprosy

The Madrid Classification

Lepromatous type (L)

Macular

Diffuse

Infiltrated

Nodular

Neuritic, pure (possibly?)

Tuberculoid type (T)

Macular (Tm)

Minor tuberculoid (micropapuloid) (Tt)

Major tuberculoid (plaques, annular lesions, etc.) (TT)

Neuritic, pure (Tn)

Indeterminate group (I)

Macular (Im)

Neuritic, pure (In)

Borderline (Dimorphous) group (B)

Infiltrated

(Others ?)

Indian Classification, 1955

Lepromatous	Intermediate	Non-lepromatous
Maculoanesthetic	Indeterminate	
Tuberculoid	Borderline	
Polyneuritic		

The Ridley–Jopling classification

based on

1) Clinical,
2) histological,
3) bacteriological and
4) immunological

5-group system:

1) Tuberculoid (TT),
2) Borderline Tuberculoid
3) Midborderline (BB),
4) Borderline Lepromatous (BL) and
5) Lepromatous (LL) [10,15]

CLINICAL FEATURES OF LEPROSY

Indeterminate Leprosy

This form of leprosy is commonly seen among school children. One to three ill-defined hypo pigmented macules size ranging from 1 to 5 cm on the outer side of the extremities, buttock, face, and trunk with thickening of nerve. Slit skin smears may not reveal acid-fast bacilli. The lesions may heal spontaneously in a majority, small proportion may end in tuberculoid or lepromatous pole depending on the immune status of the person [11,13.]

Tuberculoid Leprosy (T)

Macules or plaques, 1 to 3 in number, and their size ranging from 0.5 cm to 30 cm or more, anesthetic and are frequently associated with thickened nerve trunk. Commonest sites involved are the face, lateral or dorsal aspects of the extremities, and the buttocks. Enlargement of one or two nerves at the vicinity of the skin lesions. They are unilateral and asymmetrical. Margins are sharply defined and infiltrated, but the centre is less infiltrated. The surface is dry and sweating is lost. Slit skin smears are negative. The lepromin test is strongly positive (2+ to 3+).

Borderline Tuberculoid (BT)

This is the commonest type of leprosy characterized by well defined infiltrated plaques,3 to 10 in number with well defined and raised margin in a part of a lesion, flat or vague in another and presence of satellite lesions. Peripheral nerves are irregularly enlarged at the site of predilection in an asymmetrical pattern.

Midborderline (BB) Leprosy

It is the most unstable and uncommon form of the disease. It is characterized by erythematous infiltrated plaques, 10 to 20 in number with a punched out or annular appearance, usually bilaterally but asymmetrically distributed. The plaque is depressed in the centre and has a sharp inner edge and sloping outer border.

Nerve damage may be variable; It may be unilateral or bilateral depending on whether it evolved from the BT or the BL.[16]

Borderline Lepromatous (BL)

Macular, annular plaques or even nodular lesions may occur in BL. They are copper colored, and more infiltrated in the centre compared to the periphery. Number of lesions varies from 20 or more. Anesthesia is more in the centre than periphery. The lesions are bilateral and progresses towards symmetry. Diffuse infiltration of pinna and eyebrows are seen. Lepromin test is negative. Glove and stocking anesthesia is not present thus differentiating it from LL.

Lepromatous Leprosy:

Highly infectious form of the disease where the cell mediated immunity is lost, involves multiple organs like skin nerves, eyes, testes, adrenals,etc.Ill-defined macules which is either hypo pigmented or erythematous are present initially. The lesions are bilaterally symmetrical with smooth shiny surface. Infiltration progresses gradually leading to the development of papules, nodules, and plaques, assuming a waxy appearance, on forehead, the chin, ear lobes , forearms and dorsum of hands.

Madarosis is characteristic of lepromatous leprosy. Massive infiltration of bacilli in to the face leads to the development of leonine facies characterized by prominent ridges and furrows seen on the forehead ,thickened ears and loss of eyebrows. Nasal mucosa is involved in majority of cases, presenting with nasal stuffiness and epistaxis. Nasal secretion is highly infectious.

Corneal involvement leads to the development of ulceration, Lagophthalmos is due to the infiltration of leprosy bacilli in to the zygomatic fibers of facial nerves. Repeated orchitis in type 2 reaction causes testicular atrophy, and thereby gynaecomastia. Glomerulonephritis

occurs in type 2 reaction [17] and rarely may progress to end stage renal failure. SSS is –positive and lepromin test becomes negative .[14]

RARE VARIANTS OF LEPROSY:

1) Lucio's leprosy
2) Lazarine leprosy
3) Histoid leprosy
4) Pure neuritic
5) Localized lepromatous leprosy

Lucio's leprosy:

Is the rare form of the LL seen in Mexico. Diffuse infiltration involving the face and most of the body, giving waxy and shiny appearance to the skin, preceded by nasal congestion, epistaxis, hoarseness of voice, edema of hands. Madarosis may occur first. As the disease advances, thickening of eyelids gives sleepy or sad appearance. It is called Lepra bonito, meaning beautiful leprosy.[46]

Lucio phenomenon (lepra manchada) is a type 3 lepra reaction occurs in Mexicans with diffuse non nodular form of leprosy, seen mainly in untreated patients. [18]

Lazarine leprosy:

It is an unusual manifestation of BT Hansen's manifesting as edema, formation of blister and ulceration of skin lesions which occurs spontaneously. Predisposing factors are protein malnutrition. Pathology being accentuated hypersensitivity in type1 reaction.[19,29.]

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Histoid leprosy:

A rare variant of lepromatous leprosy, characterized by the development of multiple cutaneous or subcutaneous nodules and plaques arising on an apparently normal skin with unique morphology of the bacilli and HP.

Causes are

1) Monotherapy with Dapsone
2) Inadequate and irregular treatment
3) Drug resistance to Dapsone
4) Emergence of mutant organisms from a Dapsone – susceptible population of bacilli [21]

Clinical features:

Well demarcated nodules situated on the back, buttocks, face extremities and elbow.

Difference between lepromatous and histoid nodule

LEPROMATOUS NODULE	HISTOID NODULE
Edge, merges with the surrounding infiltrated skin.	Well demarcated, arises on a normal looking skin.

Histopathology:

Numerous spindle shaped histiocytes, arranged in a whorled pattern, loaded with bacilli. Bacilli appear to be longer with tapering ends, arranged in parallel stacks called histoid habitus. Some lesions show nests of epitheloid cells called tuberculoid contaminants.[22]

Differential diagnosis:

1) Lepromatous nodule
2) Molluscum contagiosum
3) Erythema nodosum
4) Neurofibroma

Pure neuritic:

Leprosy involving only nerves without skin lesions. Wade recognized this entity as pure neuritic form. The spectrum varies from tuberculoid to borderline lepromatous. If single nerve is grossly enlarged and lepromin test becomes positive then the spectrum is tuberculoid.. Ulnar and lateral popliteal nerves are commonly affected. However sural, superficial peroneal and greater auricular nerves are also involved. Pure neuritic form is usually a borderline tuberculoid Hansen. Follow-up of these patients reveals the development of skin lesions in 35% of cases over 3–5 years with or without treatment.

Localised Lepromatous leprosy:

This is a rare form which contains either a single nodule or multiple nodules over a particular localised area sparing the remaining parts of the body. SSS revealed higher bacterial index, while rest of the skin is negative for bacilli.

Reactions of leprosy:

Definition:

Reactions are due to acute episodes of hypersensitivity phenomenon to the antigen of M.Leprae. It occurs due to immunological instability of the host

Types

1. *Type 1 lepra reaction*, (synonyms - Coombs and Gell's type IV reaction) called delayed hypersensitivity reaction and is mediated by T lymphocytes;
2. *Type 2 lepra reaction*, (synonyms- erythema nodosum leprosum (ENL), coombs and Gell's type III hypersensitivity) mediated by humoral immunity, associated with immune complexes.
3. Lucio phenomenon or necrotic lepra reaction. Leprae bacilli invade the endothelium of blood vessels and causes necrosis of arterioles.

Type 1 reaction:

Seen commonly in the age group of 20-40yrs.Affects both the sexes equally. But male had slight preponderance of nerve damage with borderline tuberculoid. Occurs within 6 months of starting MDT in nearly 80% of cases. Seen mainly in borderline spectrum (BT, BB, BL) of patients because of their immunological instability.[23,48]

It can be either upgrading (reversal reactions) or a downgrading reaction

Precipitating factors for upgrading reactions: [24]

1) Dapsone therapy
2) pregnancy
3) vaccinations
4) post partum

Precipitating factors for down grading reactions [25]

1) infection
2) stress
3) surgery
4) pregnancy

Grading of type 1 reaction: [24]

Mild -Erythema, tenderness and swelling of a existing lesions.

Moderate-Erythematous plaques with effusion of joints and edema of limbs.

Severe – ulceration of the skin lesion , painful neuritis with paralysis.

Risk factors:

1) Enlargement of a nerve.
2) Large patch over the face
3) High bacteriological index
4) Neuritis
5) Disseminated patches involving larger areas

Clinical features:

Increased erythema and edema of pre-existing skin lesions with distinct edge. They are tender and warm. New lesions may occur but very rare. Sometimes ulceration occurs. Edema of hands and feet occurs. Acute painful neuritis is a serious complication result in sudden paralysis, e.g. foot drop, facial palsy. Rarely nerve abscess may occur.

Complications:

1) Claw hand, food drop, wrist drop and facial paralysis.
2) Ulceration of the existing lesions.

Differential diagnosis:

1) Urticaria
2) Erysipelas
3) Relapse

	Reversal reaction	Down grading reactions
Treatment	On taking treatment	Irregular treatment/defaulter
Existing skin lesion	Well defined, more prominent	Less prominent, margins undefined
New skin lesions.	No	More new lesions
Neuritis	No new nerve damage	New nerve damage occurs
BI	Decreases	Increases

Treatment:

Standard Dose schedule for field purpose (WHO 1998)

DOSE	WEEKS OF TREATMENT.
40mg daily	2weeks
30mg daily	2weeks
20mg daily	2 weeks
15 mg daily	2 weeks
10mg daily	2 weeks
5mg daily	2 weeks

Treatment of Type 1 lepra reactions [23]

1) Mild reaction

Chloroquine and aspirin

2) Severe reaction

Steroids and aspirin with or without large doses of clofazimine. splinting of affected nerves in the acute stage.

Type 2 reaction:

Also known as Roseolar leprosy, Erythema nodosum leprosum(ENL), Lepromatous lepra reaction, acute exanthem of leprosy. Occurs most commonly in lepromatous leprosy and rarely in borderline lepromatous. Seen among 20-40 years of age. Occurs after 2 to 3 years of starting multi drug therapy.

PRECIPITATING FACTORS: [26]

1) Psychological stress
2) Intercurrent infection
3) Injury, surgery
4) Drugs-dapsone, rifampicin
5) Ingestion of alcohol, vaccination

Classification of type 2 reaction: [28]

Based on the mode of onset

1)Rheumatic type-starts with fever, joint pain and later skin lesions develop.

2)Exanthematous type-fever and skin lesions develop simultaneously.

3)Mixed type-starts with fever, joint pain and skin lesions simultaneously.

Clinical features:

Most characteristic skin lesion of type 2 is ENL. They occur as painful, evanescent (lasting for 2-3 days),erythematous raised, dome shaped nodules occurs in crops in the evening (endogenous cortisol is least). Occurs on the face, arms, in a bilaterally symmetrical fashion. Recurrences occur at the same site. Papules, nodules and plaques are the classical lesions.

Clinical variants of ENL:

1) Vesicular
2) Bullous
3) Haemorrhagic
4) Pustular
5) Ulcerated
6) Erythema multiforme like [27]

SYSTEMIC MANIFESTATIONS

EYE:

Conjunctivitis, keratitis, iritis, episcleritis, and iridocyclitis and lagophthalmos occurs in type 2 reaction .

ENT:

Involves the mucous membranes of mouth, nose, resulting in rhinitis, epistaxis, ulceration and perforation of nasal septum.

MUSCULOSKELETAL:

Arthritis and myositis are the commonest manifestation. Arthritis affects knee, metacarpophalangeal, interphalangeal joints, ankle and wrist. Dactylitis occurs as painful spindle shaped swellings of the phalanges.

Lymphadenitis:

Preauricular lymphadenitis is a premonitory sign in type 2 reaction.

EPIDERMOORCHITS:

U/L or B/L, acute painful swollen testis that later leads to atrophy.

Grading of Type 2 lepra reactions: [28]

1) Mild: Temperature above 100˚F, few skin lesions on one or more extremities.

2) Moderate: Temperature up to 102˚F, numerous skin lesions in all four limbs and few on the trunk and face with vesicle and pustule.Extracutaneous signs present.

3) Severe: Temperature above 102˚F, vesiculation and postulation occurs in skin. Visceral involvement occurs.

Complication

1) Ulceration of skin lesion
2) Blindness
3) Infertility
4) Perforation of nasal septum.
5) Paralysis of involved nerves.

Treatment: [47]

1) Mild/moderate reaction

Rest + aspirin + chloroquine (when associated with bone/joint problems only)

2) Severe ENL:

Rest + analgesics + steroids/thalidomide(23)

Clofazimine in larger doses

3) Thalidomide especially in cases of chronic recurrent ENL and steroid dependence.

Type 3 lepra reaction(Lucio phenomenon)

Occurs in a non nodular form of lepromatous leprosy. Presented as a diffuse shiny infiltrative lesions in Mexicans called Lucio Leprosy. Seen especially in untreated patients. Painful, ill defined red-bluish plaque surrounded by an erythematous halo present on the extremities. Later the lesions become purpuric, centre of the lesion undergoes necrosis and forms a black crust. Rarely bullous lesions which burst open to form a deep ulcer with jagged edges are seen. Patients remain a febrile throughout the course of reaction. [18]

Complication:

1) Secondary bacterial infection and
2) Secondary amyloidosis.

DIFFERENTIAL DIAGNOSIS OF LEPROSY

1) Macular Skin Lesion :

Vitiligo

Hypo pigmented or depigmented macules with normal sensation, no peripheral nerve thickening. Depigmentation usually does not occur in leprosy.

Pityriasis versicolor-

Hypopigmented macules usually smaller than leprosy macule, associated with fine branny scales. Sensation is intact, there is no hair loss and and no peripheral nerve thickening.

Pityriasis Alba

Asymptomatic, round-to-oval, ill-to-well defined hypo pigmented macules on the face with fine scales. Seen in children, mimicking inderminate leprosy. No impairment of sensation, loss of sweat and hair. Self resolution occurs.

Nevusachromicus

Usually presents at birth, circumscribed hypopigmented macule with serrated margins**.** Does not disappear on diascopy.

Nevus anemicus

A vascular anomaly which produces hypopigmented macule and it disappears on diascopy.

Post inflammatory hypopigmented macule

There is a history of primary dermatosis.

Lichen sclerosus et atrophicus

There is hypo pigmented mildly atrophic plaque. Sensation is intact. Peripheral nerves are normal. Histopathology is diagnostic.

Differential Diagnosis of Infiltrated Skin Lesions

Lupus Vulgaris-

Lupus in early stage mimics tuberculoid leprosy. Infiltrated, well defined plaque, with a tendency to heal with scarring. They heal at one edge and spread at another, which is characteristic of lupus vulgaris whereas central healing is observed in leprosy.

Psoriasis

An erythematous infiltrated scaly plaque which mimics tuberculoid or borderline leprosy in reaction. Auspitz sign is positive. Lesions situated on the extensor aspects of the extremities, scalp, and trunk associated with nail pits.

Sarcoidosis

Infiltrated plaque of sarcoidosis resembles tuberculoid or borderline leprosy. The sensations of the plaque and peripheral nerves remain normal. Histopathology is diagnostic.

Dermatophyte Infections (Ringworm or tinea corporis):

It mimics tuberculoid or borderline leprosy. The inflamed borders are studded with papulovesicles. There is central clearing and peripheral extension of scales.

Granuloma Annulare

Children and young adults are most commonly affected and it is a differential diagnosis for tuberculoid or borderline leprosy. Symptomatic

papules and nodules in an annular configuration. Histopathology reveals a necrobiotic pallisading granuloma which is characteristic of GA.

Syphilis

Annular lesions of secondary syphilis resemble leprosy but the involvement of the palms and soles, with generalized lymphadenopathy, positive serology (TPHA or VDRL) differentiates it from leprosy.[29]

Differential Diagnosis for enlarged nerves are

1) Leprosy
2) Neurofibromatosis
3) Charcot–Marie–Tooth disease (types 1 and 3)
4) Refsum's disease
6) Amyloidosis
5) Sarcoidosis

Nerves apparently enlarged conditions are

1) Cyst
2) Tuberculous abscess
3) Post-traumatic

DIAGNOSIS OF LEPROSY.

SLIT SKIN SMEAR: [9]

Skin is cleaned with ether and a portion is gripped between thumb and index finger and blanch it .With a small – bladed scalpel an incision is made between the fingers about 5 mm long and 3 mm deep, with the pressure of the fingers being maintained .The blade is then turned at right angles to the cut and the wound is scraped several times in the same direction so that issue fluid and pulp collects on one side of the blade, and smeared on a glass slide. Two or more smears can be made on one slide. The sites should be noted, so that the same sites can be used for successive sets of smears during the course the treatment.

Slides with smears on them should not be exposed to sunlight, dust; extremes of temperature and humidity. This may interfere with the capacity of the bacilli, to take up carbol fuchsin

Nerve biopsy:

Is essential for pure neural leprosy. A thickened sensory nerve is suitable, such as supra-orbital branch of the 5th cranial nerve, supra clavicular nerve, great auricular nerve in the neck, sural nerve at the back of the leg. These nerves do not contain motor fibers, therefore no risk of motor damage.

HISTAMINE TEST:

When histamine is injected intra dermally in to normal skin causes capillary dilatation, seen as a bright red flare known as histamine flare. This is due to axon reflex within the dermal nerves.

SWEATING(NINHYDRIN

For testing the integrity of dermal nerves. Anhidrosis is characteristic of skin lesions in tuberculoid leprosy. Injecting 0.2ml of a 1 in 1000 solution of pilocarpine nitrate in to the tested lesion, paint the injection site with tincture of iodine, and then dust with starch powder.

Sweating causes blue discoloration of the powder. Absence of blue discoloration indicates that sweating is absent due to dermal nerve damage. [30]

Bacteriological index

Density of bacilli in smears is known as bacteriological index. It includes both solid staining and fragmented bacilli.

:

0: No bacilli in 100 oil immersion fields

1+: 1–10 bacilli in 100 oil immersion fields

2+: 1–10 bacilli in 10 oil immersion fields

3+: 1–10 bacilli in an average oil immersion field

4+: 10–100 bacilli in an average oil immersion field

5+: 100-1000 bacilli in an average oil immersion field

6+: >1000 bacilli in an average oil immersion field.

The Morphological Index (MI)

Calculated as the percentage of solid staining bacilli in a smear after examining 200 bacilli . Solid staining bacilli indicates only the living bacilli.[31]

WHO disability grading, 1998

Hands and feet [9]

Grade 0: No anesthesia, no visible deformity or damage

Grade 1: Anesthesia present, but no visible deformity or damage

Grade 2: Visible deformity or damage present.

Each hands or foot assessed and graded separately. Damage includes ulceration, shortening, disorganization, stiffness and loss of part or all of the hand or foot.

Eyes

Grade 0: No eye problem due to leprosy, no evidence of visual loss

Grade 1: Eye problems due to leprosy present, but vision not severely affected as a result of these (vision: 6/60 or better; can count fingers at 6m)

Grade 2: Severe visual impairment (vision: worse than 6/60;inability to count fingers at 6 m)

Eye problems due to leprosy include lagophthalmos, iridocyclitis, and corneal opacities.

OCULAR LEPROSY:

Eyes will be affected in leprosy in 4 ways:

1) Abnormal exposure of the eyes secondary to involvement of 5th & 7th cranial nerves

2) Infiltration of the eyes secondary to direct invasion of lepra bacilli.

3) Inflammation of the eyes secondary to infiltration.

4) Complication of eyes secondary to involvement of surrounding tissues, eyelids, lacrimal glands.[34]

In tuberculoid patients, eye is affected when there is an inflammatory patch over the branches of the facial nerve which innervate the Orbicularis oculi muscle responsible for closing the eyes, usually unilateral.

Lepromatous Leprosy patient are susceptible for all complications of eye, but slower and symmetrical.

Madarosis:

Loss of eyebrows and eyelashes is called madarosis due to direct invasion of M.leprae which destroys the roots of the cilia.

Facial nerve abnormalities:

Zygomatic branch of facial nerve which innervates the orbicularis occuli muscle is very much susceptible to leprosy bacilli results in lagophthalmos. As a result of loss of blinking, poor spread of tear and increased evaporation of tears leads to corneal ulceration. Secondary infection leads to endophthalmitis, resulting in blindness. Primary aim of treatment is to keep the cornea moist by giving tear substitutes. Physiotherapy is given by teaching the patient to close his eyes forcefully many times a day, thereby strengthening the remaining functioning muscle fibers. Lateral tarsorraphy is the surgical procedure for lagophthalmos. Systemic treatment is indicated.

INVASION OF THE EYE:

Mainly by blood spread. The pathological lesions are in the conjunctiva, cornea, sclera, iris and ciliary body.

Leprous keratitis:

Bacilli migrate along the myelinated corneal nerves to form microscopic lepromata or beads in to the corneal stroma .Lesions found on superolateral aspect of cornea beneath the upper eyelid. Inflammation leads to vascularisation of corneal stroma and opacification resembling trachoma pannus. Difference being more deeper and superolateral in leprosy whereas superficial and centre in trachoma.

Infiltration of the iris and ciliary bod

Occurs via blood spread. Appearance of small lepromata near the pupillary margin .They appear as round, white and shiny called iris pearls. Presence of M.leprae in iris and ciliary body cause iridocyclitis, which is the most common cause of irreversible blindness in leprosy.(35)

Deformity:

Is defined as a visible impairment and the consequences of the impairment.

Impairment :

Defined as the physiological, anatomic or physiological losses resulting from a disease.

1) Primary impairment

2) Secondary impairments. [40]

Types of Deformities

1. Specific deformities: due to infiltration with M. leprae.
2. Paralytic deformities: due to motor nerve damage.
3. Anesthetic deformities: secondary to impairment because of damage to motor nerves.

Specific and paralytic deformities are examples of primary impairment. Anesthetic deformities are examples of secondary impairments.[36]

Specific deformities:

Occurs in lepromatous patient .face is commonly involved.

1) Loss of eyebrows,

2) Premature senility of the face

3) Nasal deformities

4) Deformities of external ear.

5) Terminal phalange resorption

6) Banana fingers.

Specific deformity occurring in ENL:

Hand is commonly affected with

1) Intrinsic plus finger deformity (swan-neck deformity)

2) Twisted fingers

3) Non paralytic clawing.

Paralytic deformities:

Commonly hands, less frequently in feet. Face occurs in borderline and pure neuritic types of leprosy.

1) Claw-finger,

2) Claw thumb deformities.

3) Wrist drop

4) Lagophthalmos

5) Facial palsy.

Anaesthetic deformities:

Feet is the commonly affected part with

1) Plantar ulceration

2) Shortening of digits.

3) Scarring of the sole

4) Mutilation

5) Tarsal disorganization.

6) Corneal ulceration.

7) Blindness. [37]

Disability prevention:

Have three aims

1)Prevent nerve damage and consequent nerve function deficit, deformities, disabilities, and disablement of the patients.

2)Avoid secondary impairments (like wounds, ulcers, skin cracks, scar contractures, joint stiffness and serious eye problems).

3).Recognize secondary impairments as early as possible, when they have occurred, manage them properly so that they do not become complicated and need expert advice and intervention, which is not easily available.[38]

Disability prevention practices by affected persons have four Components:

1) Skin care,

2) Wound care,

3) Joint care, and

4) Eye care:

1) Skin care practice

It is essential to prevent a breach of the insensitive skin of the hands and feet. To achieve this, patients were taught to use the anesthetic hand carefully, avoiding injuries, uses protective footwear to avoid injury to the insensitive feet.

Hydration of the dry anesthetic skin of hands and feet and to prevent skin cracks. Health education to inspect his hands and feet at least once a day to identify any injury and takes treatment to get it healed at the earliest.

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2) Wound care:

For gaping wounds patient needs medical advice without delay as they require immediate suturing,

Small non gaping wounds will be washed with soap and water, it should be covered clean and dry. If it is not healed within 72 hours then seek medical advice.

Persistent swelling with local warmth should alert the person of a possible closed injury, and medical advice must be sought without any delay. Till then, the part must be covered with a bulky compression bandage, rested, and kept elevated.

4)**Joint care:**

Aim is to keep the joints in paralytic deformities mobile and free from contractures. Massaging the digits using a vegetable oil makes the skin soft. Repeated passive stretching of the bent digits helps in straightening. Active exercises to extend the finger joints.Oil massaging and stretching exercises are done daily for 10-15 minutes.

If finger contractures have already occurred, should be opened out gradually without damaging the contracted structures using serial splinting.

Plantar Ulceration (trophic ulcer)

Commonest problem seen in leprosy found in about 10% of patients. They are seen mostly (80% of cases) in ball of the foot (medial part) in the metatarsophalangeal (MTP). 5% to 10% in the mid-lateral part of the sole underlying cubo-metatarsal joint and another, 5% to 10% in the heel.

Etiology and Pathogenesis

Three different reasons

1. **loss of sensation**-

   The insensitive sole is injured due to nails, thorns, etc the wound is neglected because of loss of sensation , and it gets infected and develops into a ulcer.

2. **Through a fissure in the skin**:

   The dry, anesthetic skin develops fissures or fine cracks .in corns and callosities and later ulcer.

3.Motor palsy:(accounts 90% cause)

Forefoot is subjected to stress and strain during walking, and these are contracted by muscles of the foot. When these muscles are paralyzed, some areas are overloaded, causes the tissues to break down, leading to ulceration.

Stages in the pathogenesis of ulcer:

1**) Stage of 'threatened ulceration'** in which the stressed subcutaneous tissues present in the ball of the foot undergoes traumatic inflammation. This may be identified by dorsal puffiness and splaying of the great toe and 2nd toe. The foot should be rested in a splint and kept elevated for 72 hours. No weight-bearing on the affected foot.

2) Stage of 'concealed ulceration' (necrosis blister) :Continued walking causes necrosis and liquefaction of the subcutaneous tissue, and the liquefied material reaches the surface and presents as a blister. The blister is padded well or, if there is a danger of breaking open, it is snipped and covered with Vaseline gauze, and a below-the-knee plaster cast applied.

3) The third stage of 'open ulceration' where the necrosis blister breaks open and exposed to the external world.

Types of ulcers

1) acute,
2) chronic,
3) complicated and
4) recurrent

1) Acute ulcers:

They are frankly infected, purulent, covered with slough and are acutely inflamed.

2) Chronic ulcer

Indolent ulcers with heaped up hyperkeratosis edges, having a serosanguineous discharge and covered with pale granulation tissue is called chronic ulcer. Simple chronic ulcer do not present any complication. Complicated chronic ulcers presents with involvement of deeper structures like an underlying bone, joint, and tendon sheath and pseudoepitheliomatous hyperplasia, malignant transformation (usually squamous cell carcinoma) and life-threatening infections like gas gangrene, septicemia or tetanus.

Management:

Acute ulcer needs 1) Absolute bed rest.

2) Elevate foot

3) Eusol bath, irrigation, dressing

4) Limit surgery to drainage procedures

5) Antibiotics if needed

Chronic ulcer

Simple ulcer can be managed by

Scraping floor of ulcer,

Vaseline gauze dressing

Below knee POP cast or bulky dressing

Protective footwear + foot-care training

Complicated Ulcer can be managed by

Debridement

Physiological rest by below knee POP.

Protective footwear on POP removal

Skin graft large ulcers

Recurrent ulcers can be managed by

Scar revision using excision and suture, local flaps, distant flaps, free flaps, footwear modification or corrective surgery and eradication of infection.

Feet with plantar sensory loss and no muscle paralysis:

Footwear have a tough outer sole, resist penetration by thorns, nails, glass pieces, etc, should not damage the foot, so no nails, uppers/straps and buckles .Automobile tire pieces are used as outer soles .

Insensitive feet having intrinsic muscle paralysis:

These feet requires a resilient, non collapsing and shock-absorbing insole insert that will reduce the stresses generated during walking .Microcellular rubber (MCR) of 10° to 20 used.

Dense plantar scars due to previous ulceration:

Modification of the footwear by adding a metatarsal bar obliquely across the sole about 25 mm proximal to the metatarsal heads or inserting a medial arch [41]

In some feet the pressure will still be rather high at vulnerable sites.

Maximum reduction of pressure is achieved by molding the insole thereby spreading the load of body weight over the entire plantar surface .

Foot Care Practice:

Foot is soaked in a water for 15 to 20 minutes and then the sole is rubbed against a coarse surface or pumice stone. which removes the heaped up layers of keratin, then neem oil is smeared over the sole and the dorsum without drying the foot. Soaking softens the skin, rubbing removes the keratin layers, and the oil layer traps moisture and keeps the skin hydrated.

Other precautions are

1. Protective MCR footwear even in a home.
2. Advice to minimize walking (e.g. use bicycle where possible).
3. To avoid walking long distances.
4. Find safe limits' of walking.
5. Have to walk a long distance, interrupt the same with periods of rest.

National leprosy control programme: [42]

The government of India in 1943 appointed the Health Survey and Development committee with Sir Joseph Bhore as chairman. The committee was put forward for the development of a national programme of health services for the country**.** The committee's recommendations on leprosy were creation of provincial leprosy organizations, increase the existing provision for institutional treatment of out-patients and in-patients and financial help to voluntary organizations engaged in anti-leprosy work.

Based on this recommendation, NLCP a central aided programme was **s**tarted in 1955. Its aim was to achieve control of leprosy through early detection of cases and on dapsone domiciliary treatment through vertical units. It was done by implementation of Survey Education and Treatment (SET) activities.

The goal of Government of India was to eradicate leprosy by 2000 and appointed a working group in 1980. Working group submitted its report in 1982 and recommended multi drug therapy.

The control programme was revised as National leprosy Eradication Programme(NLEP) in 1983.The districts were covered in a phased manner and all the districts in the country were covered by the year 1996. Main objective was to reduce case load to 1 or less than 1/10,000 population.

In 1991, World Health Assembly resolved to eliminate leprosy at a global level by the year 2000. The first phase of the World Bank supported National Leprosy Elimination Project was launched in 1993 and completed in March 2000.

NLEP introduced the Modified Leprosy Elimination Campaign (MLEC) activities in the year 1997-98. Five such campaigns were conducted up to the year 2004.

The 2nd phase of the World Bank supported National Leprosy Elimination Project was launched in 2001 and completed in December 2004. During this phase the responsibilities of NLEP were decentralized from the centre to the states. Leprosy services were also integrated with the General Health Care System from the vertical system.

A system of monitoring of the programme was started in the form of Leprosy Elimination Monitoring (LEM) exercise jointly by Govt of India with WHO in 2002-2004. During the year 2003-2004, component of validation of case diagnosis was introduced.

Leprosy was declared eliminated as a public health problem in India at National level in the month of December 2005. From then onwards the program continues as an integrated part of general health service.

Components of the current programme:

1) Decentralized integrated leprosy services through general health care system .

2) Capacity building of all general health services functionaries.

3) Intensified information ,education and communication.

4) Prevention of disability and medical rehabilitation.

5) Intensified monitoring and supervision. [50]

Major initiatives:

1) Main focus given to new case detection which is the main indicator for programme monitoring than prevalence.

2) Treatment completion rate has been taken as an important indicator.

3) Major emphasis on disability prevention and medical rehabilitation service to leprosy affected patients.

a) Dressing materials, supportive medicines and ulcer kits are provided to leprosy affected persons .

b) Micro–cellular rubber foot is provided for protection of insensitive feet.

c) An amount of Rs.5000/ is provided as incentive to each leprosy patient who is below poverty line, undergoing reconstructive surgery in the identified institutions to compensate for loss of wages

4) ASHAs have been involved in bringing out suspected leprosy cases from their villages for diagnosis and treatment at PHC and follow up of confirmed cases for their treatment completion. To motivate ASHA ,incentive money given.

i) Confirmed case brought by them will be paid RS 100.

ii) On completion of full course of treatment of the case within specified time they were paid Rs 200 for PB cases and 400 for multibacillary cases.

5) Disability prevention and medical rehabilitation(DPMR).

6) Intensive IEC campaign with a theme ‘Towards Leprosy free India’ has been carried out towards further reduction of leprosy burden in the community,early reporting of cases and their treatment completion. Awareness generation activities are carried out through mass media and local media.

Disability prevention and medical rehabilitation(DPMR):

1) Implementation of DPMR activities as per guidelines and reporting its outcome e.g. treatment of leprosy reaction, ulcers, physiotheraphy.etc.

2) Integrating DPMR services: convergence of NLEP services in to National Rural Health Mission facilities.

3) To develop a referral system to provide prevention of disability services to all leprosy disabled persons in an integrated set-up.

Activities carried out in different setups:

Sub centre:

Self care advice and monitoring activities are carried on. Disability and reaction should be referred to PHC.

Primary health centre :

Reactions can be managed if possible or referred. Identification of patients need reconstructive surgeries, foot wear and advice of wound care. Complicated ulcers, eye problems, reactions not able to manage should be referred.

District hospitals:

Should manage complicated ulcers and lepra reactions. Patient need reconstructive surgeries should be referred to district nucleus.

MULTIDRUG THERAPY:

Objectives are

a) To interrupt transmission of the infection in the community as rapidly as possible with the bactericidal drugs.

b) Early detection and treatment of cases to prevent deformities.

c) To prevent drug resistance.

Rationale for MDT:

Newly diagnosed ,untreated lepromatous case may harbor 10^{10} - 10^{11} viable M.leprae. Chance of resistance to a single drug is only 1in 10^{6}-10^{7} bacilli. Then the chance of resistance to 2 drugs is 10^{12}-10^{14},which is less than the no of bacilli found at the time of diagnosis. The chance of bacilli resistant to all three drugs would be then nil.

WHO recommendations:

1) MB leprosy-a person having 6 or more skin lesions and /or more than one nerve involvement. Treatment given 12 months ,and should be completed within 18 months.

1) Rifampicin - 600mg once monthly given under supervision.

2) Dapsone - 100mg daily ,self administered.

3) Clofazimine -300 mg once monthly supervised ,and 50 mg daily, self administered.

2) PB leprosy-person having 1-5 skin lesions and/or only one nerve involvement. Treatment is given for 6 months and should be completed within 9 months.

1) Rifampicin - 600mg once monthly given under supervision.

2) Dapsone - 100mg daily, self administered.

Materials and methods

Study population

Entire population of Alanganallur block . Permission from ethical committee of the hospital obtained for the study.

Methodology:

The survey was conducted with the help of 25 Accredited Social Health Activist (ASHA) appointed for that block by Director of Leprosy and their field work was supervised by Health inspectors, Non medical supervisors and Investigator. These ASHA workers were trained to undertake house to house survey in their villages to identify suspected cases of leprosy, leprosy-related disability and complications and old cases using verbal questionnaire. I the Investigator, examined these suspects to filter out non leprosy cases and the leprosy cases were confirmed by a thorough clinical examination, slit skin smear and biopsy when necessary.

Soft ware used is SPSS Chi-square test.

Training to ASHA:

Initially 2 days comprehensive training programme was conducted for these ASHA workers. During the training, audiovisuals and clinical case description which includes all aspects of leprosy, disability ,and clinical features differentiating it from non leprosy cases was taught.

Inclusion criteria:

All confirmed cases of leprosy among the suspected cases in Alanganallur block were included in the study. The cases were categorized as new cases, old cases and defaulters.

New case of leprosy is defined as a person with at least one cardinal feature of leprosy but who had not taken treatment for the leprosy so far, irrespective of duration of the disease. The cardinal features of leprosy are hypo pigmented or reddish skin patches and with definite signs of anesthesia, nerve involvement and slit skin smear positive for acid fast bacilli. This includes previously undiagnosed cases of Hansen's with symptoms of the disease of longer duration as well as cases of recent onset.

Old case is defined as a person with signs and symptoms of leprosy, who had either taken treatment fully or in part .

Defaulter is defined as a person who is not taking treatment continuously for 3 months in paucibacillary group and six months in multibacillary group.

Exclusion criteria:

Non leprosy cases in Alanganallur block and leprosy patients from outside Alanganallur block.

Observation and Results:

Total population enumerated in Alanganallur block was 94544.Total number of ASHA's involved in this study were 25. Population surveyed door to door by ASHA's from 1.4.2013-31.3.2014 was 77585(82%) .

Table 1: **DEMOGRAPHY**

Variables	**Alanganallur**
Total population enumerated	94544
Total number of PHC	4
Total population surveyed	77585(82%)
Total number of suspects	894(1.15%)
Number of suspects examined.	803(90%)

Among 77,585(82%) people screened, the number of suspected cases found were 894 (1.15%). Among this suspected people 91 (10%)were lost for follow-up. Total number of suspected cases examined were 803(90%).

Table 2: **Distribution of cases in the survey**

Variables	Number	Percentage
Examined suspects	803	
Leprosy cases	83	10%
Non Leprosy Cases	720	90%

Among the suspects examined (803), the leprosy cases constituted 83 (10%) and non leprosy cases were 720 (90%).

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Table 3: **Distribution of leprosy cases**

Variables	Number
Total Leprosy Cases In Survey	83
New Leprosy Cases	12 (15%)
Old Case On Treatment	6 (7%)
Released from treatment (RFT) Cases	60 (72%)
Defaulters	5 (6%)

Among the 83 cases of leprosy in the survey, 12 (15%) were new leprosy cases. Old cases on treatment were 6 (7%). RFT includes 60 cases (72%) and defaulters were 5 (6%).

Table 4: **Distribution of non leprosy cases**

S.No.	Diagnosis	Count	Percentage
1	Tinea versicolor	401	54%
2	Tinea corporis	153	21%
3	Pityriasis alba	40	5%
4	Eczema	34	5%
5	Vitiligo	28	4%
6	Polymorphic light eruption	16	2%
7	Post Inflammatory hypopigmentation	11	1%
8	Allergic contact dermatitis	10	1%
9	Nevus Achromicus	8	1%
10	Miscellaneous	37	5%

Among Non Leprosy Cases Tinea Versicolor contributes 54% and Tinea Corporis 21%, Pityriasis Alba 5%, Vitiligo 4%, Polymorphic Light Eruption, 2% post inflammatory hypopigmentation, nevus achromicus contributes less than 1%.Miscellaneous cases include keloid, furuncle, neurofibroma and insect bite allergy.(table 4).

Table 5: Classification of New leprosy cases.

S.No.	Variables	Numbers	Percentage
1	Total new leprosy cases	12	
2	Paucibacillary cases	7	58%
3	Multibacillary cases	5	42%

Among 12 newly diagnosed leprosy cases, PB contributes 7 (58%) and MB contributes 5 cases (42%).

Table 6: **Age wise distribution**:

Variables	Number	Percentage
Child	2	17%
Adult	10	83%

Among 12 new cases, 2 (17%) cases were children (under 15 years) and 10 (83%) cases were adults.

Table 7: Age wise distribution

	Paucibacillary		Multibacillary	
Age	**No.**	**%**	**No.**	**%**
< 20 Years	2	29%	0	0%
20-40 Years	3	43%	0	0%
41-60 Years	2	29%	5	100%
> 60 Years	0	0%	0	0%

Among PB the commonest age group affected is 20-40 years which contributes 43%. Among MB the commonest age group affected is 41-60 years. All the cases belong to this age group.

Table 8: Sex wise distribution

	Paucibacillary	Multibacillary
Sex	**No. (%)**	**No. %**
Male	4 (57%)	3 (60%)
Female	1 (14%)	2 (40%)
Male Child	1 (14%)	0 (0%)
Female Child	1 (14%)	0 (0%)
TOTAL	7	5

Males accounted for 57% of PB type, ,females 14%and children 28%. Male: female ratio among MB cases was 6:4.There were no childhood MB cases.

Table 9: Spectrum of Disease among Male and Female

Type of Leprosy	Male	%	Female	%
TT	1	8%	1	8%
BT	7	58%	2	17%
BB	0	0%	0	0%
BL	0	0%	1	8%
LL	0	0%	0	0%

BT Hansen is the most common in male and female which constitutes about 58% and 17% respectively. TT Hansen constitutes 8% among male and female. A single case of BL Hansen was seen in a female. No case of lepromatous leprosy was found.

Among 12 cases 6 had nerve involvement in which 4 were males and 2 females.

Table10: **Nerve Involvement in New Leprosy cases**

Type of Leprosy	Male	%	Female	%
Nerve Involvement among Male and Female	4	67%	2	33%

Nerve involvement is more common in male than in females.

Table 11: **Nerve involvement in correlation with the spectrum of leprosy.**

Type of Leprosy	Male	%	Female	%	Total %
BT	4	67%	1	16.5%	83.5%
BL	0	0%	1	16.5%	16.5%

Among BT Hansen 67% of male and 16.5% of female had nerve involvement. Among BL Hansen one female had nerve involvement.

Among 6 patients, 4 had single nerve involvement and 2 had multiple nerve involvement. Among 6 cases with nerve involvement Ulnar nerve is more commonly involved than other nerves. .

Table 12: **Duration and site of skin lesion**

S.NO	PATCH	SITE	DURATION
1	Hypopigmented	Knee	2 months
2	Hypopigmented	Right forearm	1 months
3	Hypopigmented	Trunk	1-2 years
4	Hypopigmented	Face,Upper limb,Lower limb	4 years
5	Hypopigmented	Elbow	6 month
6	Hypopigmented	Back,trunk	3-4 years
7	Hypopigmented	Right leg	3 months
8	Hypopigmented	Trunk	2-3 years
9	Hypopigmented	Left arm	2 months
10	Erythematous	Right thigh	1 year
11	Hypopigmented	Back	2 years
12	Hypopigmented	Right face	1 year

Table (12) shows the duration of the lesion, in that nearly 7 cases had 1 year and above 1year duration.

Deformity

Among 12 cases, 2 (17%) had deformity.

Reaction:

Among 12 case of leprosy, 1 male had type 1 reaction. This contributes 8% of newly diagnosed cases.

National Leprosy Eradication Programme(NLEP) evaluation indicators:

Annual new case detection rate(ANCDR)

$$= \frac{\text{No. of New cases detected during a specified period of time}}{\text{Population in that area during a specified period.}} \times 100000$$

Our survey detected 12 new leprosy cases. So ANCDR is

$$\frac{12}{77{,}585} \times 100000 = 15.5$$

Prevalence:

$$\frac{\text{No. of balance cases under treatment as on 31st March}}{\text{Population as on 31st March}} \times 10000$$

$$\frac{10}{77{,}585} \times 10000 = 1.3$$

Proportion of multibacillary(MB) cases

$$\frac{\text{Number of MB cases detected in a year}}{\text{Total new cases detected in a year}} \times 100$$

$$\frac{5}{12} \times 100 = 42$$

Proportion of child cases

$$\frac{\text{Number of child cases (< 15 years) detected in a year}}{\text{Total new cases detected in a year}} \times 100$$

$$\frac{2}{12} \times 100 = 17$$

Proportion of female cases:

$$\frac{\text{Number of female cases detected in a year}}{\text{Total new cases detected in a year}} \times 100$$

$$\frac{3}{12} \times 100 = 25$$

$$\text{Proportion of deformity cases} = \frac{\text{Number of deformity cases detected in a year}}{\text{Total new cases detected in a year}} \times 100$$

$$\frac{2}{12} \times 100 = 17$$

Table 13: **Comparison of Survey Indicators with NLEP, DPMR- Alanganallur Block (2012-2013)**

Variables	Survey Data	NLEP.DPMR (1.04.2012-31.03.2013)	p value
ANCDR/100000	**15.5**	**11**	**0.441 NS**
Prevalence rate/ 10000	**1.3**	**1**	**1.000 NS**
Proportion of MB Cases	**42**	**75**	**0.020 SIG**
Proportion of PB Cases	**58**	**25**	**0.003 SIG**
Proportion of children	**17**	**8.3**	**0.137 NS**
Proportion of female	**25**	**42**	**0.097 NS**

DPMR- District Progressive Monthly Report. There is a statistically significant p value seen for proportion of multibacillary(MB) cases and paucibacillary(PB) cases. NS indicates not significant. NLEP- National Leprosy Eradication Programme .

Table-14 : **Comparison of Survey Indicators with NLEP**

DPMR Alanganallur Block(2013-2014)

Variables	Survey Data .	NLEP DPMR (1.04.2013-31.03.2014)	p value
ANCDR/100000	**15**	**13.8**	**1.000 NS**
Prevalence rate	**1.3**	**0.92**	**1.000NS**
Proportion of MB Cases	**42**	**40**	**0.956 NS**
Proportion of PB Cases	**58**	**60**	**0.976 NS**
Proportion of children	**17**	**20**	**0.784 NS**
Proportion of female	**25**	**47**	**0.036 NS**

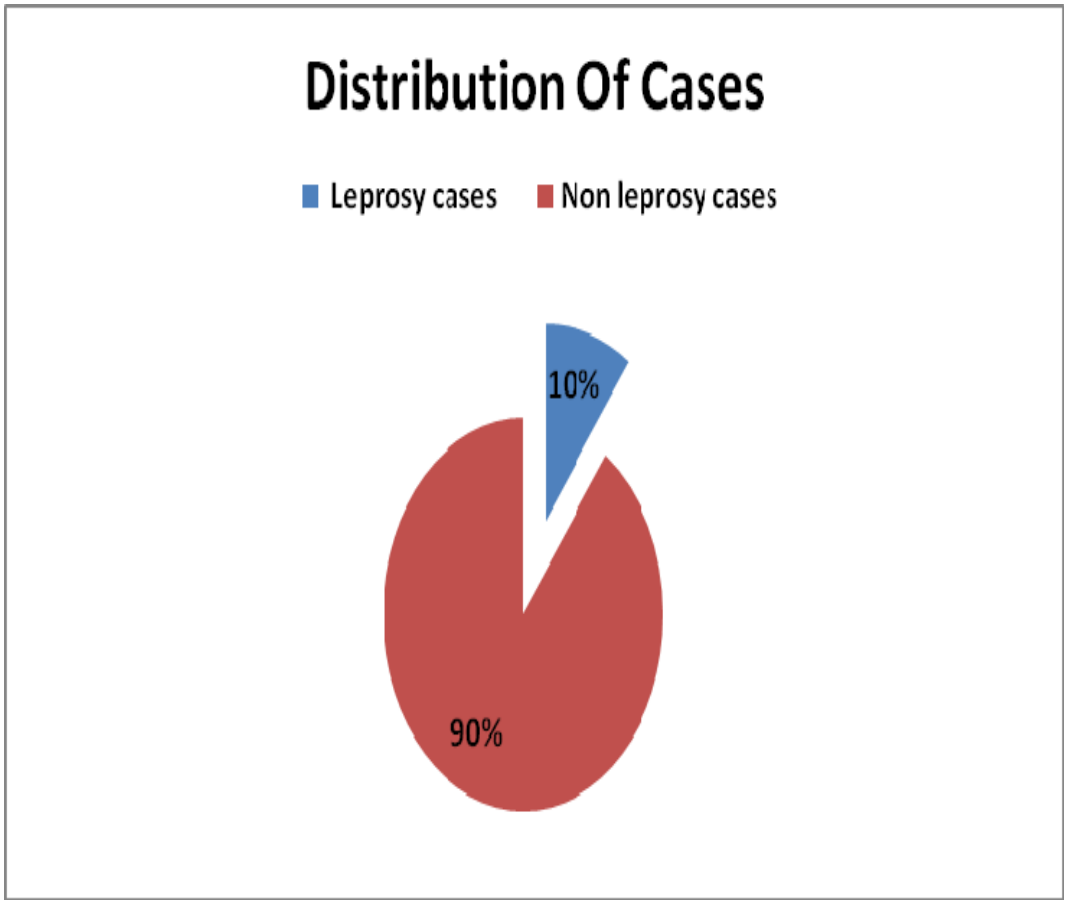
DPMR- District Progressive Monthly Report. On comparison the p value is not statistically significant on comparing the survey data with the NLEP DPMR 2013-2014.

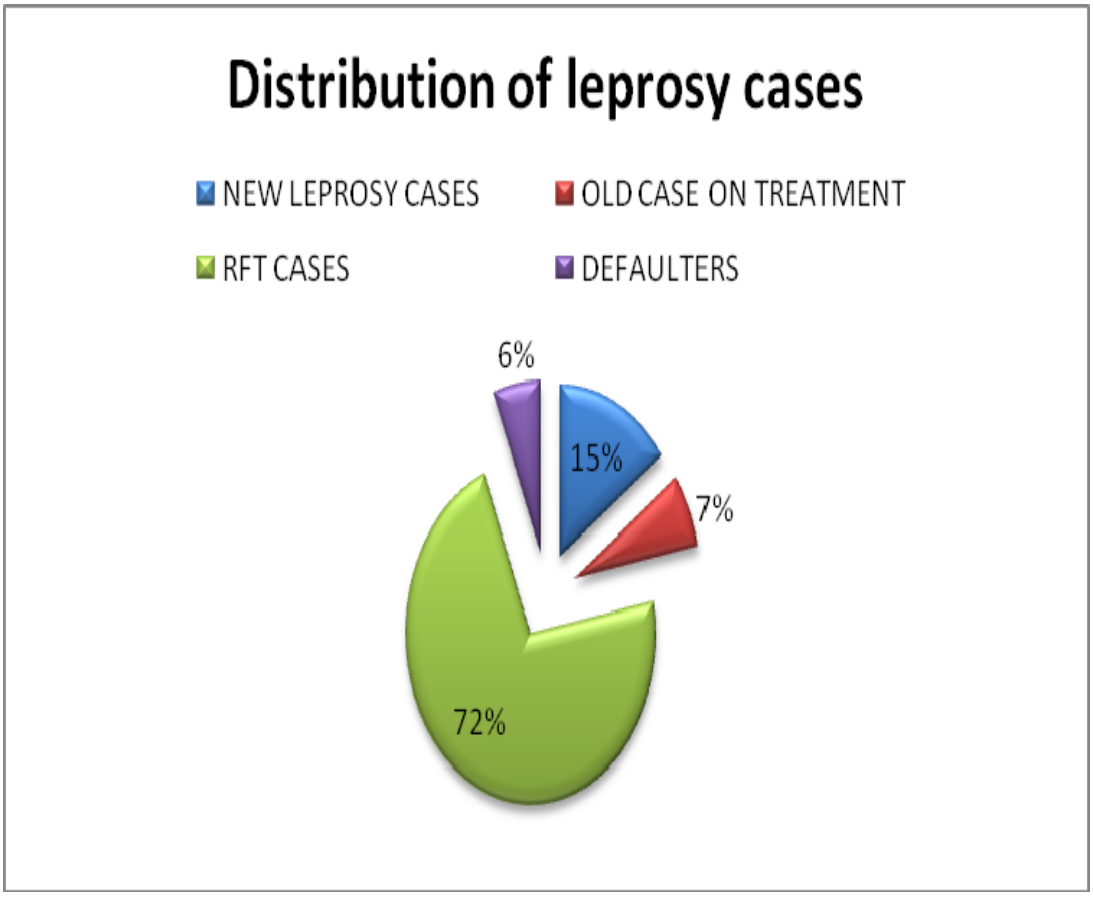
Table15: **Comparison of Survey Indicators with NLEP**

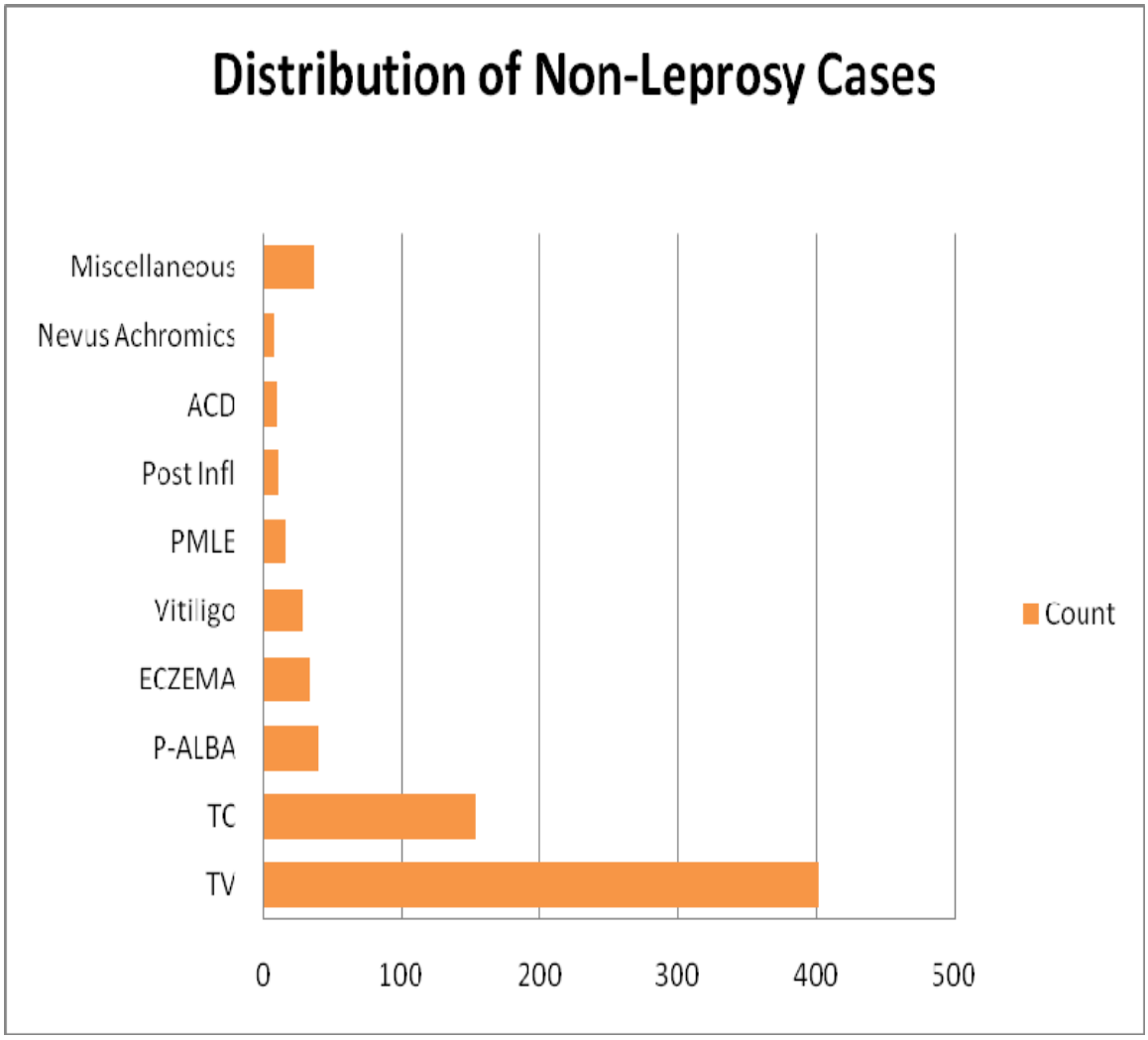
Tamil Nadu Government Data(2013-2014)

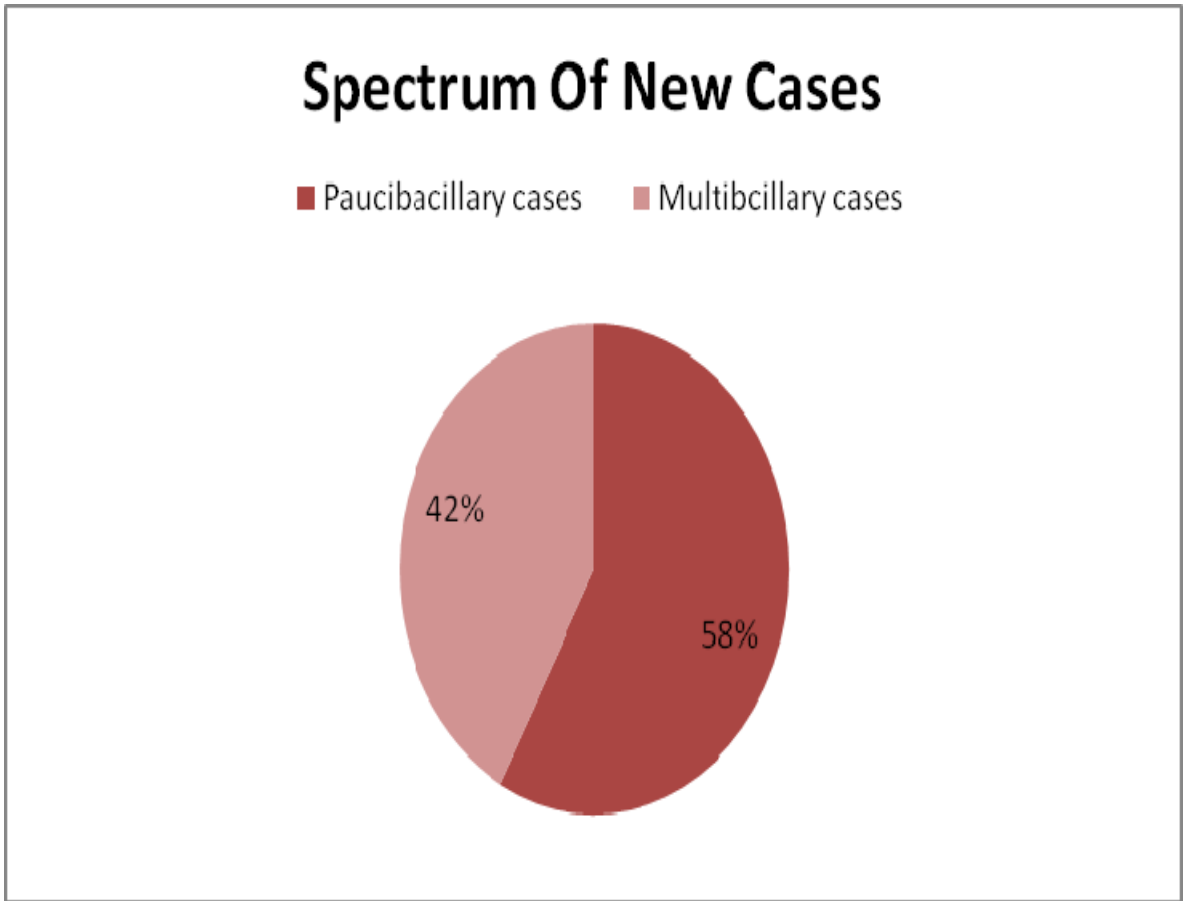
	Survey Data	**NLEP, TamilNadu (1.04.2013-31.03.2014)**	**p value**
ANCDR/100000	**15**	**5.057**	**0.044 Sig**
Prevalence rate	**1.3**	**0.4**	**1.000 NS**
Proportion of MB Cases	**42**	**54**	**0.377 NS**
Proportion of PB Cases	**58**	**46**	**0.404 NS**
Proportion of children	**17**	**9**	**0.205 NS**
Proportion of female	**25**	**29**	**0.742 NS**

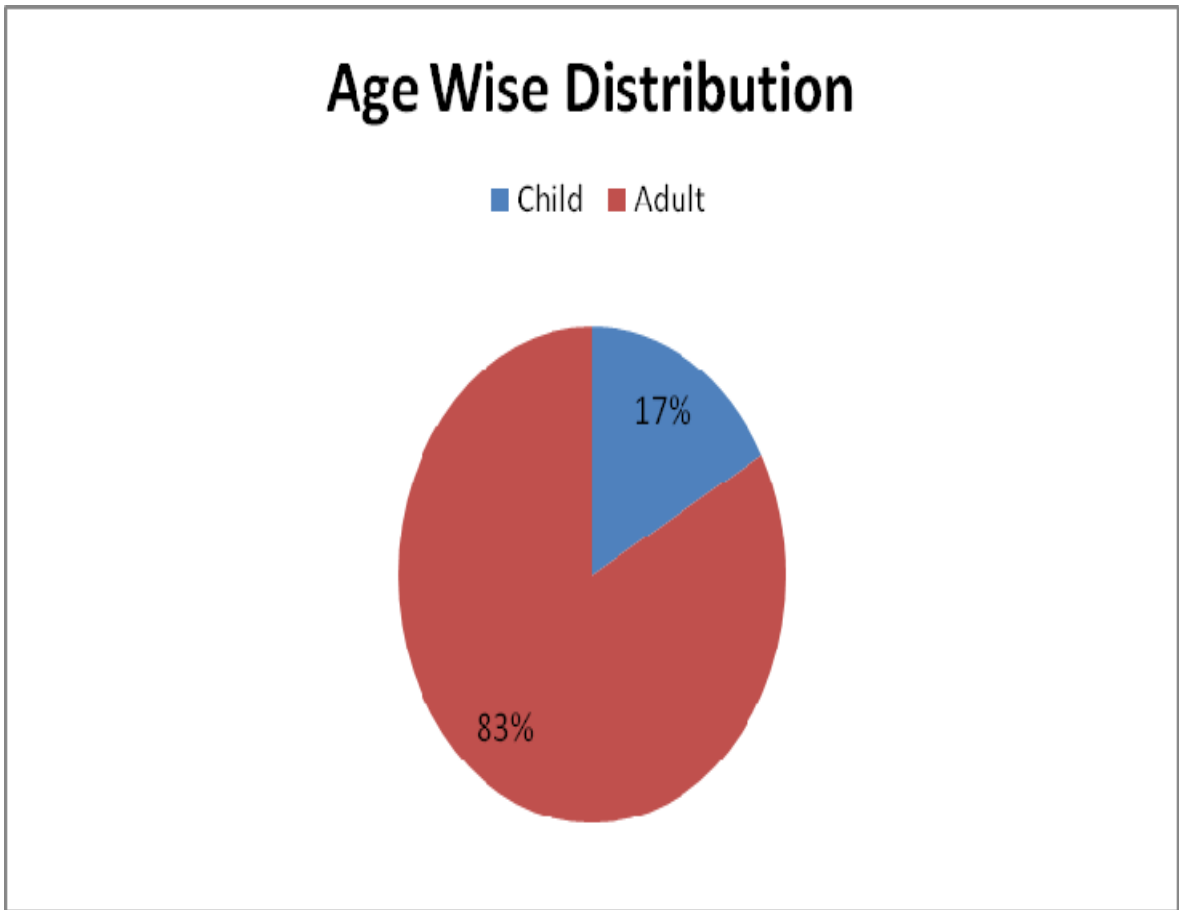
There is statistically significant p value seen in ANCDR of survey data compared with NLEP Tamilnadu-2013-2014

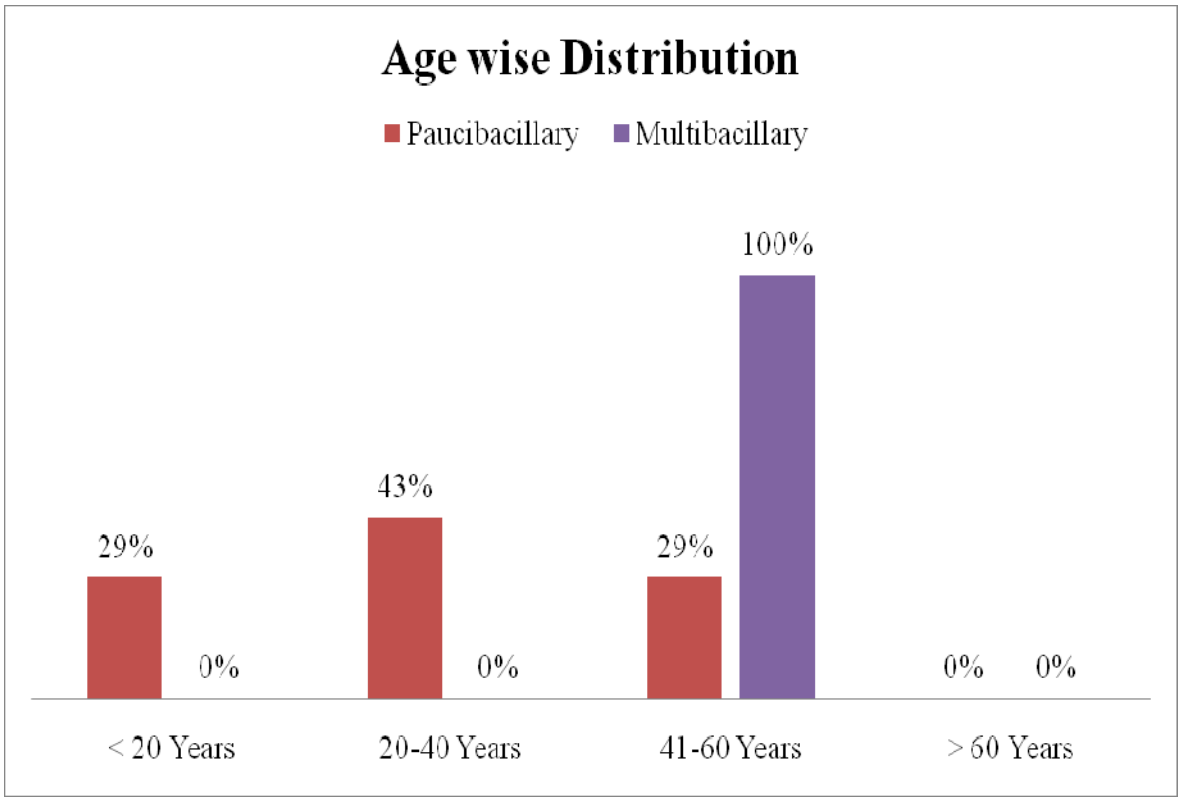

Distribution Of Cases
Leprosy cases
Non leprosy cases
10%
90%

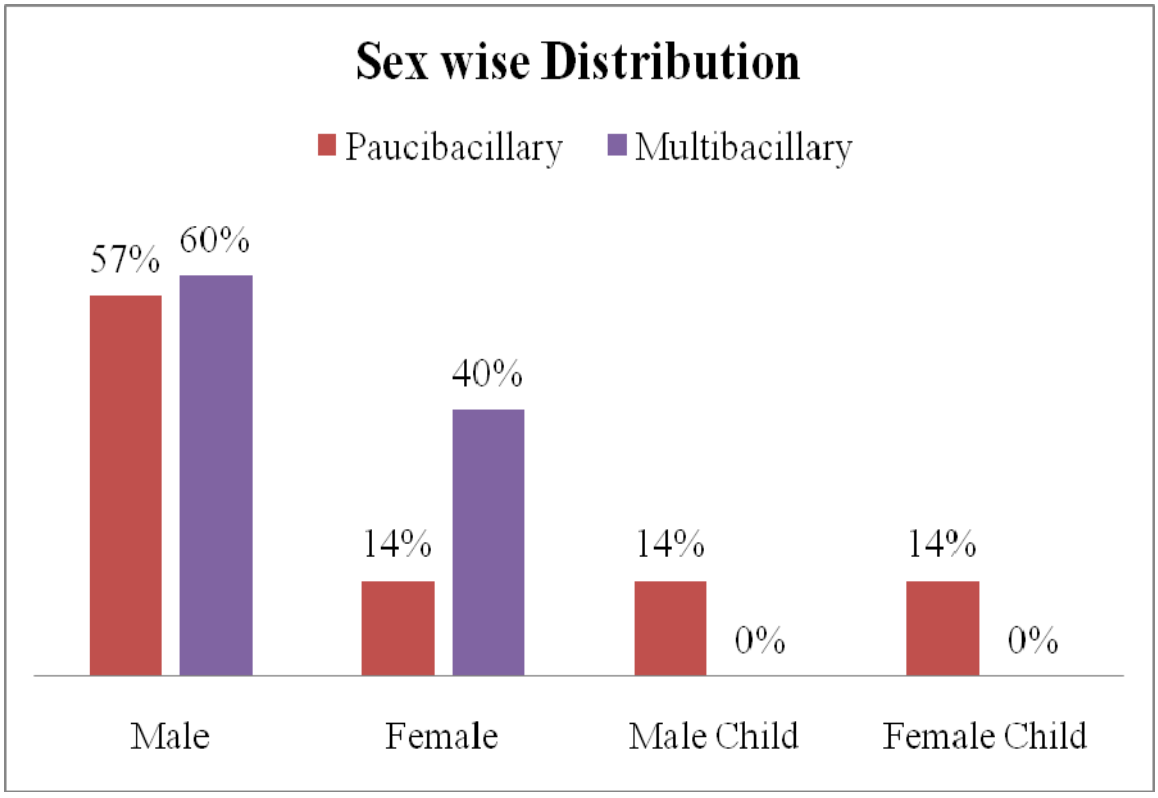

Distribution of leprosy cases
NEW LEPROSY CASES
OLD CASE ON TREATMENT
RFT CASES
DEFAULTERS
6%
15%
7%
72%

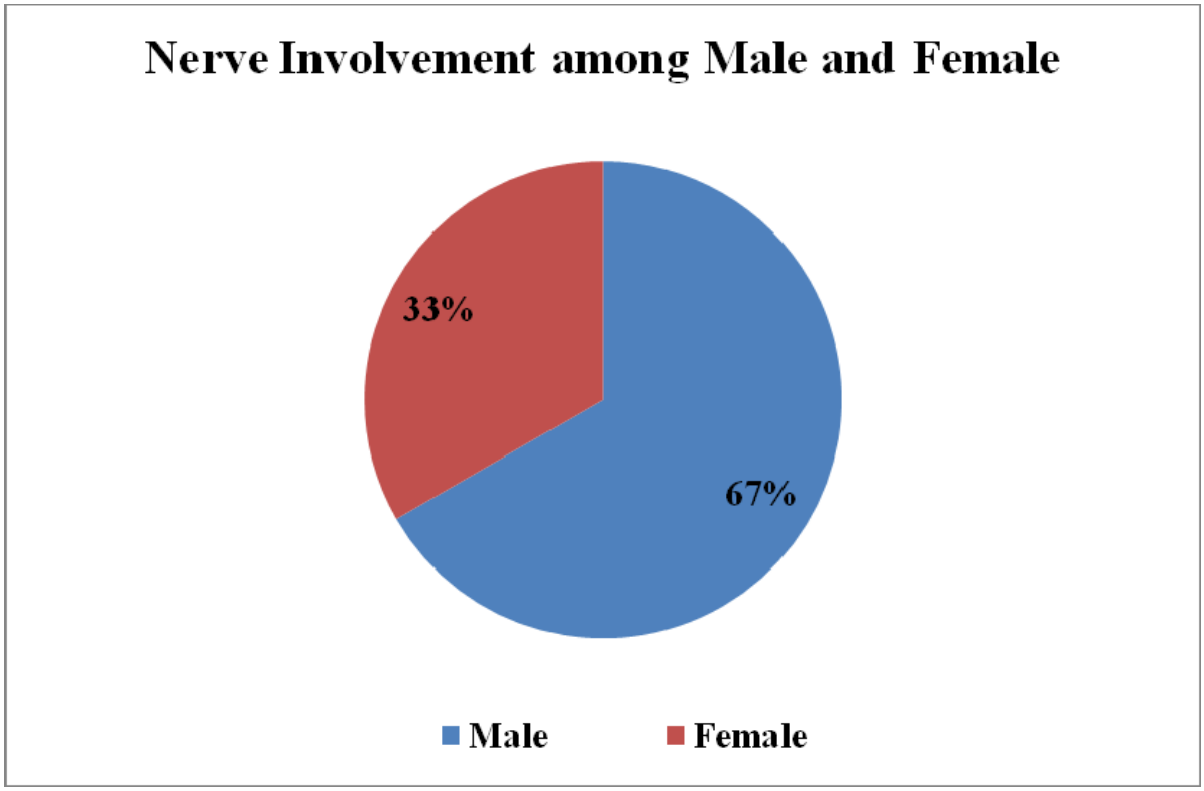

Distribution of Non-Leprosy Cases
Miscellaneous
Nevus Achromics
ACD
Post Infl
PMLE
Vitiligo
ECZEMA
P-ALBA
TC
TV
0
100
200
300
400
500
Count

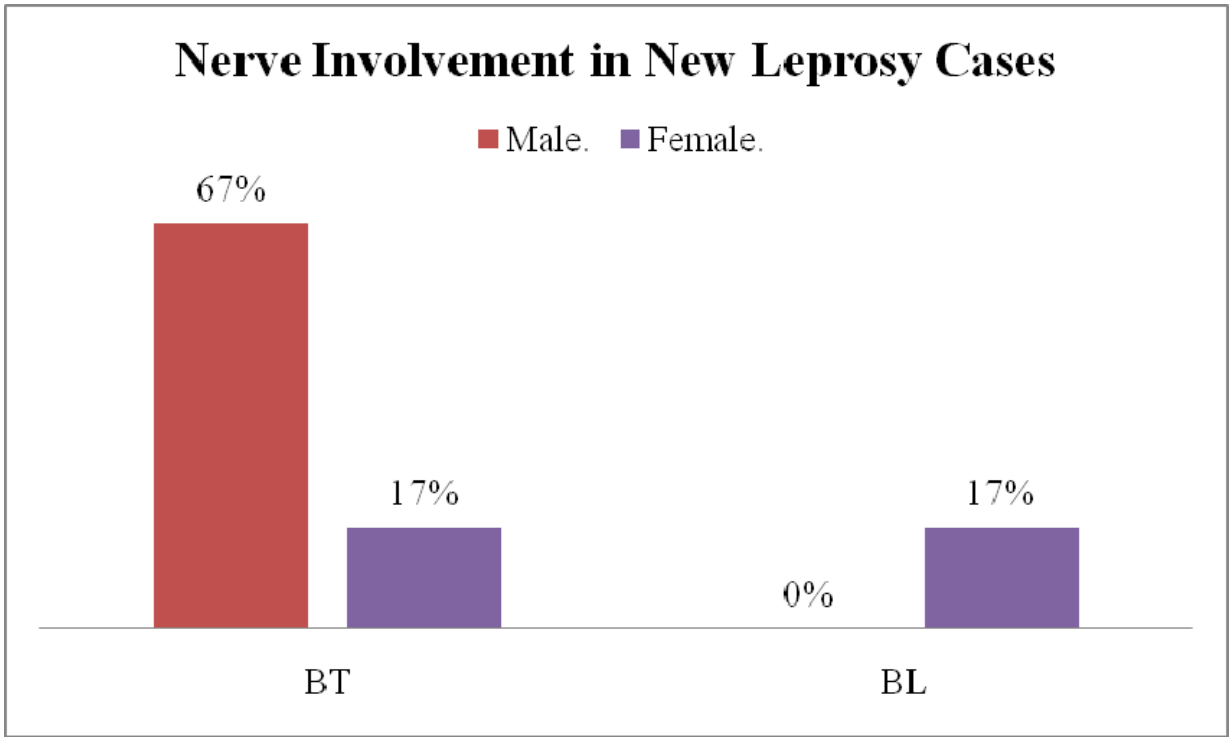

Spectrum Of New Cases
Paucibacillary cases
Multibcillary cases
42%
58%

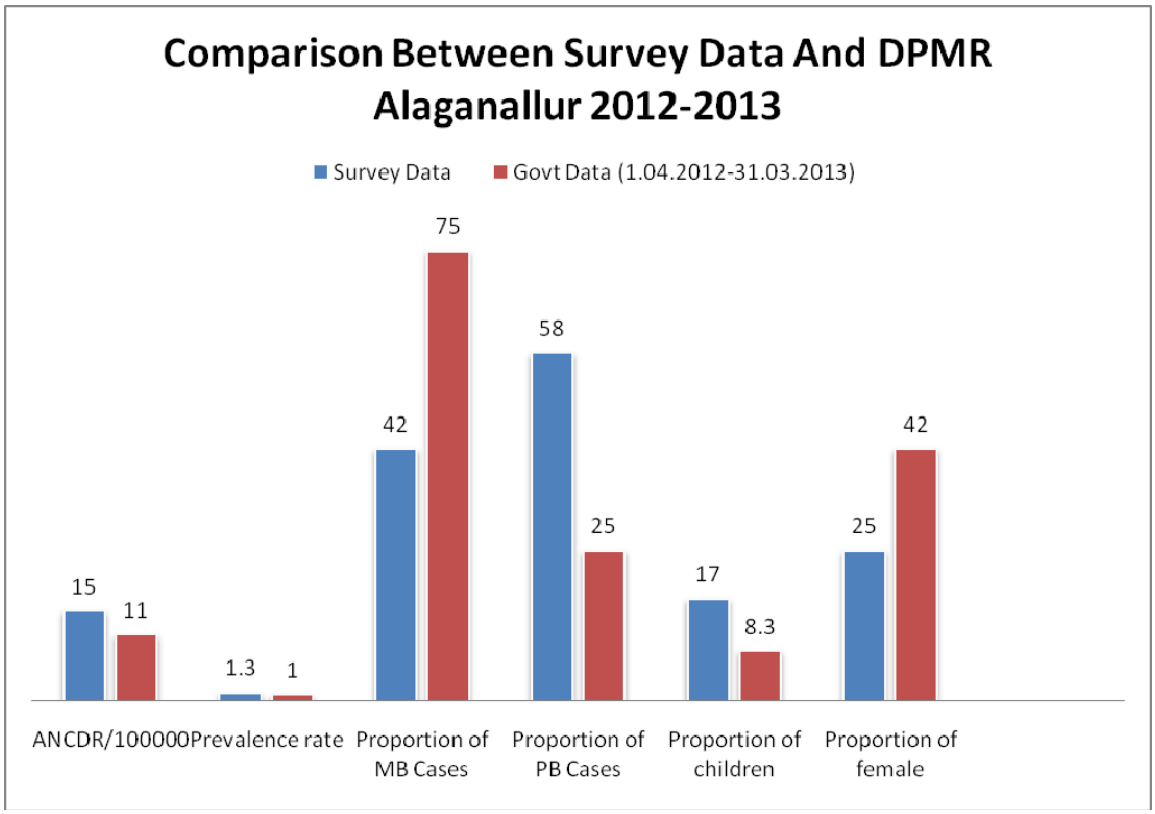

Age Wise Distribution
Child
Adult
17%
83%

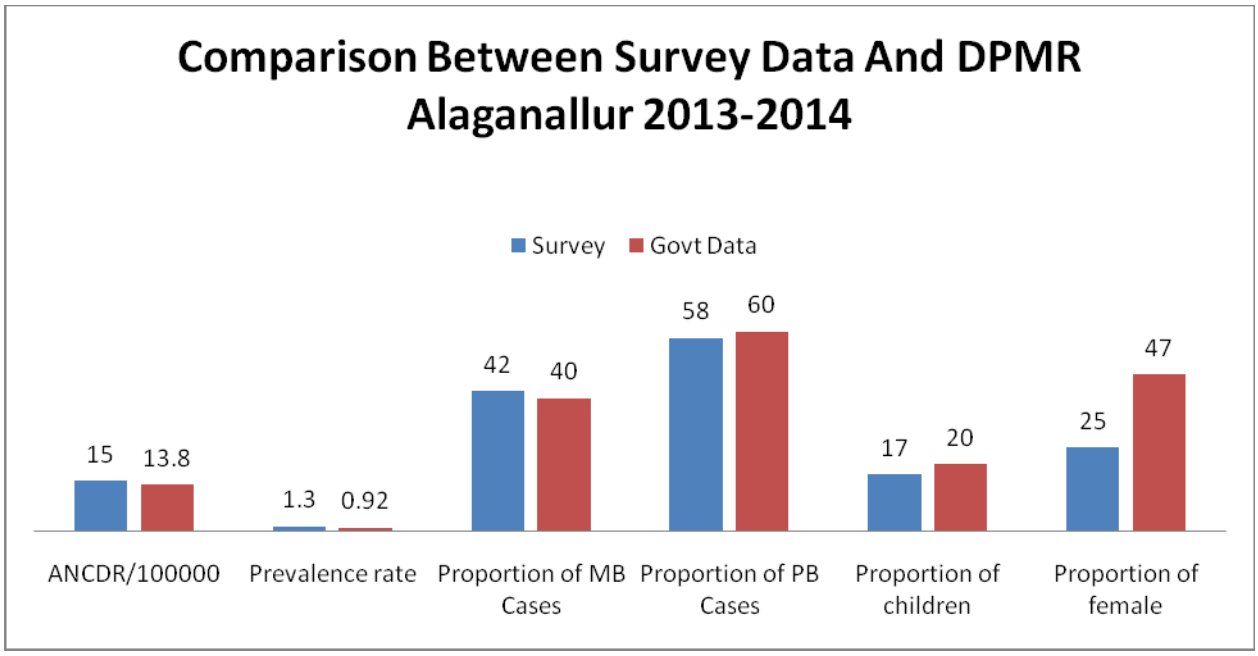

Age wise Distribution
Paucibacillary
Multibacillary
29%
0%
43%
0%
29%
100%
0%
0%
< 20 Years
20-40 Years
41-60 Years
> 60 Years

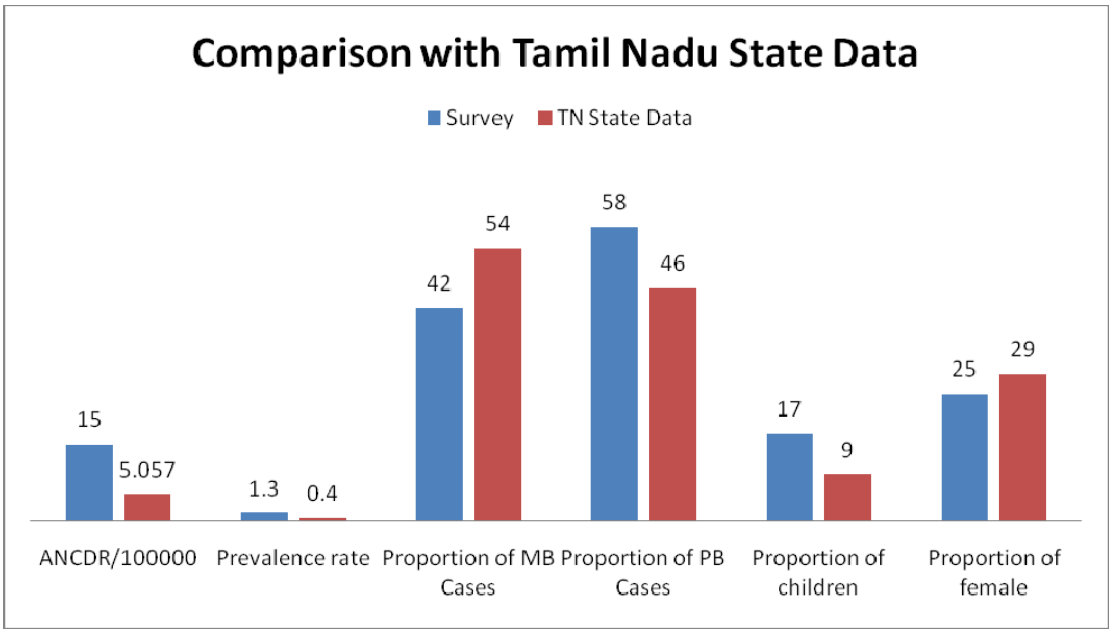

Sex wise Distribution
Paucibacillary
Multibacillary
57%
60%
14%
40%
14%
0%
14%
0%
Male
Female
Male Child
Female Child


Nerve Involvement among Male and Female
33%
67%
Male
Female


Nerve Involvement in New Leprosy Cases
Male.
Female.
67%
17%
0%
17%
BT
BL


Comparison Between Survey Data And DPMR Alaganallur 2012-2013
Survey Data
Govt Data (1.04.2012-31.03.2013)
15
11
1.3
1
42
75
58
25
17
8.3
25
42
ANCDR/100000Prevalence rate
Proportion of MB Cases
Proportion of PB Cases
Proportion of children
Proportion of female


Comparison Between Survey Data And DPMR Alaganallur 2013-2014
Survey
Govt Data
15
13.8
1.3
0.92
42
40
58
60
17
20
25
47
ANCDR/100000
Prevalence rate
Proportion of MB Cases
Proportion of PB Cases
Proportion of children
Proportion of female


Comparison with Tamil Nadu State Data
Survey
TN State Data
15
5.057
1.3
0.4
42
54
58
46
17
9
25
29
ANCDR/100000
Prevalence rate
Proportion of MB Cases
Proportion of PB Cases
Proportion of children
Proportion of female

DISCUSSION

DEMOGRAPHY

Alanganallur is a Block Primary Health centre (PHC) in Madurai district which includes 4 PHCS with the enumerated population of 94544. Population surveyed door to door from 1.4.2013-31.3.2014 includes 77585(82%).

The total number of persons examined in our study were 77585 which was 82% of the enumerated population which is higher compared to a similar study undertaken by Shetty et al [51] where the number of people surveyed were 27998 which was 69% of the enumerated population. This shows more population was covered by our survey team.

The number of suspects detected by ASHA's in our study was 894(11.5%) compared to 514(18%) of Shetty et al study.[51.]This is lower in our survey, the reason being low prevalence of leprosy in Alanganallur compared to their surveyed district which was Karjat district of Maharashtra.

90% of the suspects were screened by our team whereas 28% were screened by Shetty et al.[51] This shows 3 fold increase in the sensitivity of screening in our study.

Incidence or Annual New case detection rate (ANCDR)

The overall ANCDR of leprosy cases observed in our study was 15 per 1, 00,000 population. This is lower than the ANCDR, in the study conducted by Shetty et al.[51] which was 140/1,00,000. Alanganallur is a high prevalent area in Madurai district of Tamil Nadu but its prevalence is low compared to their surveyed district Karjat in Maharastra.

Our study is compared with the Tamil Nadu State Govt National Leprosy Eradication Programme (NLEP) monthly progress report 2013-2014 data [43] according to which the ANCDR is 5 per 1,00,000. This shows a threefold increase in ANCDR in our study.

Our study is also compared with NLEP District Progressive Monthly Report (DPMR) Alanganallur block April 2012-to-2013 [44]

where the ANCDR was 11.This shows only slight increase in ANCDR in our survey, which is not stastically significant.

But actually in our survey , nearly 60% of the Cases (table 12) have 1 or more than 1 year duration. So if active surveillance had been carried out earlier, all these cases would have been detected and treated earlier thus preventing the deformity .The ANCDR would have also been increased in the year (2012-2013). **This highlights the importance of active surveillance in detecting leprosy cases earlier.**

Our survey is also compared with the ANCDR of NLEP District Progressive Monthly Report (DPMR) of the same block for the period from April 2013-to-March 2014, where it was 13.8 per 100000 population which is nearly similar to our survey.[45]

But actually this includes the cases detected in our survey along with the voluntarily reported cases which was only 4 in number. Hence our survey cases constitute 75% of ANCDR of Alaganallur Block. **This again emphasis the importance of active surveillance in detecting leprosy cases.**

PREVALENCE

The prevalence of leprosy cases observed as on 31st March 2014 in our study was 1.3 compared with the NLEP Tamil Nadu Govt data [43] where it was 0.4. This revealed three times higher prevalence in our study.

The prevalence in our study is also compared with the prevalence of NLEP, DPMR Alanganallur block data for past 2 years, that is 2012-2013 and 2013-2014 which was 1 and 0.92 respectively which is slightly lower than our study. [44,45.]

Prevalence is not a good indicator for use in the programme because it indicates only the magnitude of the problem, i.e. the total cases registered and undergoing treatment at a point of time. However it is useful for assessing multidrug therapy requirement and also reflects efficiency in patient management.

Proportion of child cases:

The proportion of child cases in our study was 17% compared to NLEP Tamil Nadu State [43] where it was 9%. This is also compared with DPMR Alanganallur block data for past 2 years where it was 8 (2012-2013) [44] and 20 (2013-2014).[45] This shows increase in child proportion in our study.

This warrants the health functionaries that there is higher transmission of leprosy in that block and need continuous monitoring and contact survey.

The proportion of children among new cases in our study is 17% compared to 13% by Shetty et al.[51] This is higher in our study. This again indicates more active transmission of infection in the block.

AGEWISE DISTRIBUTION:

In our study, more number of cases was seen in the 41-60 years age groups. This is comparable to the increasing ANCDR from the lowest of 0.96 to 20.72 at the age of 60 years and above as observed in the study conducted by Anil Kumar et al.[52]

Among PB the commonest age group affected is 20-40 years which contributes 43%. Among MB the commonest age group affected is 41-60 years and all the cases belong to this age group.

SEX

The proportion of female cases in our study was 25% and is slightly lower in our study compared to the NLEP Tamil Nadu State data[43S] which was 29% and is not statistically significant.

Our study is also compared with the DPMR Alanganallur.

Block data 2013-2014 [45] where they were 47%. This shows decrease in female proportion in our survey.

The proportion of female cases in the Shetty et al [51] study was 13%, which is low compared to our study. The higher proportion of female case detection in our study reflects the adequate access of females in Alanganallur by our survey team.

PROPORTION OF MULTIBACILLARY CASES(MB):

The proportion of MB cases in our study was 42 which was lower than the NLEP Tamil Nadu state government data [43] where it was 54 which is stastically significant.

Our study is also compared with DPMR of Alanganallur block for the year 2012-2013 and 2013-2014, which was 75 and 40 respectively.[44,45.]

DPMR data for the year 2014 includes our survey cases too. **This highlights the early diagnosis of paucibacillary cases in our survey and relative reduction in the proportion of multibacillary cases in 2014.**

This again emphasizes the need for active surveillance in detecting leprosy cases and thereby reducing the morbidity of the disease.

MB cases contributes 5(42%) out of 12 cases, compared to 45% cases in the study by Shetty et al [51] which was comparable to our study.

DEFORMITY RATE:

Proportion of deformity cases in NLEP Tamil Nadu data [43] is 8% where as in our survey it is 17%. This is higher than the state data. This may be due to the high endemicity and actual reporting in our survey.

The number of people with deformity constitutes 17% in our study which is slightly lower than that in the study by Shetty et al (20%)

In our survey 10% of suspected cases could not be traced for examination by the investigator. Thus there is a possibility of detection of one more case if all the left over 91 cases were screened.

Summary

- Our survey detected 83 leprosy cases out of 77,584 population during the year 2013-2014.
- 12 were new leprosy cases.
- 6 cases were already on treatment
- 60 cases were released from treatment, 5 cases were defaulters
- 12 new cases include 7 PB and 5 MB cases.
- Among PB 2 were children and 10 were adults.
- It includes 2 TT, 9 BT and 1 BL Hansen's.
- 6 cases had nerve involvement. Ulnar nerve is commonly involved.
- 2 cases had deformity and 1 case had Type-1 reaction.
- The prevalence of the survey was 1.3.
- The ANCDR of the survey was 15.5 per 1,00,000 population.
- Proportion of MB and PB cases were 42 and 58 respectively.

- Proportion of child cases was 17.
- Proportion of female cases was 25.
- Disability rate was 17.

CONCLUSION

There is a large backlog in the detection of leprosy cases in the endemic areas. There are also problems faced by patients in accessing the state health care facilities. This warrants the need for active surveillance in the community to attain the actual elimination.

However it may be difficult for the government to implement active surveillance as a part of National Leprosy Eradication Programme due to financial concern. At least active surveillance needs to be reintroduced in a high endemic areas to reduce the case load and thereby attaining the actual elimination. Alternate strategies must be considered to bring out the undetected cases in the community. Information, Education and Communication activities should be strengthened to increase the level of awareness in the community and motivate the people to reach the health system.

It is necessary to ensure capacity building among primary health care staff and enforce the accountability by strict supervision.

CONCLUSION

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BIBLOGRAPHY

1. Central leprosy division –NLEP Monthly progress report for the year 2012 2013.Director General of Health services, New Delhi.
2. 496 Arch Pathol Lab Med—Vol 124, April 2000. **Armauer Hansen**—Jay. A Portrait in **History**. The Legacy of **Armauer Hansen**. Venita Jay, MD, FRCPC.
3. Van Brakel WH, de Soldenhoff, Mc Dougall Ac. The allocation of leprosy patients into paucibacillary and mltibacillary groups for multidrug therapy, taking into account the number of body areas affected by skin, or skin and nerve lesions. Lepr Rev 1992; 63::231-45
4. ILEP. The interpretation of epidermiological indicators in leprosy Techincal Bulletin 2001; ILEP, London, 3.
5. Gelber RH. Leprosy (Hansen's disease). In: Harrison's Principles of Internal Medicine, 15th edn, volume 1. Braunwald E, Hauser SL, Fauci AS et al (Eds). McGraw Hill (Pub), New Delhi, 2003; 1035-40.
6. Word Health Organization. Microbiology of M. leprae. 2009; www.who.int/microbiology/en/index.html
7. Nayar S.Narayanan JS, Job CK. Histopathological study of early skin lesions in leprosy. Arch Path 1972; 94: 199-204.
8. Levers textbook of Histopathology David E.Elder page no 188to 215

9. Rober C.Hastings. Textbook of leprosy. 1st edition. New York, Longman Group Ltd, 1989.
10. Jopling W.H.Lepr Rev 1959; 30: 194-196
11. Sardana K. A Study of leprosy in children from a tertiary pediatric hospital in India. Lepr Rev 2006; 77: 100-02
12. Pfaltzgraff RE. What is the actual male/female ratio in leprosy patiets. Lepr Rev 2003; 73: 180-81
13. Krishnamoorthy KV, Desikan KV. Indeterminate leprosy in an infant. Lepr Rev 2006; 77: 377-80
14. Pfaltzgraff RE, Ramu G. Clinical leprosy. In; Hastings RC, Diltor VAO, editors. Leprosy. 2nd edn. Edinburgh: Churchill Livingstonel 1994; 237-86
15. Kaviraj Kunjlal English translation of Sushrat Samhita in 3 volumes. Calcutta. 1907, 1911 and 1916. Quoted by Dharmendra. In: classification of leprosy.
16. Noordecn S K. Evolution of tuberculoid leprosy in a community. Lepr India 1975; 47: 85-93.
17. Kiloze AM, Ramos-Caro FA. Visceral leprosy. Int J Dermatol 2000; 39: 641-58.
18. Saoji V, Salodkar A. Lucio leprosy with Licio Phenomenon. Indian J Lepr 2001; 73: 267-72

19. Thappa DM. What is lazarine leprosy? Is it a separate entity? Indian J Lepr 2005; 77: 179-81

20. Nanda S. Bansal S, Grover C, et al. Lazarine leprosy-revisited? Indian J Lepr 2004; 76: 351-54.

21. Price EW, Fitzhebert H. Histoid variety of lepromatious leprosy. Int J Lepr 1966; 34:367-74

22. Desikan KV, lyer CG. Histoid variety of lepromatous leprosy. A histopathologic study. Int J Lepr, 1972; 40- 149-56

23. R.G. Valia and Ameet R. Valia, Bhalani [ed]. IADVL Textbook of Dermatology, 3rd edition. Mumbai, Bhalani publishing house, 2008.

24. Ridley. D.S. Lepr Rev 1982; 53: 19-26

25. Waters MFR. Int J Lepr 1971; 39: 417-428.

26. Kumar B, Dogra S, karu.I, Epidemiological characteristics of Leprosy reactions: 15 years experiences from North India. Int J Lepr Other Mycobact Dis 2004; 72:125-133

27. Petro T.S. Bullous type of reaction mimicking pemphigus in lepromatous leprosy. Ind. J. Lepr 1996; 68: 179-181.

28. Ramu. G and dharmendra in leprosy vol 1; 1978

29. Bruns T, Breathanach S. Cox N. Griffiths C. Rook's textbook of dermatology 7th edn, Massachusetts, USA. Blackwell Publishing Company, 2004.
30. Sehgal VN. Significance of local sweat response in the diagnosis of leprosy. Dermatological 1974; 148(4): 217-23
31. Ridley DS. Therapeutic trials in leprosy using serial biopsies. Lepr Rev 1958; 29: 45-82.
32. Pearson JHM, Ross WF. Nerve involvement in leprosy-pathology, differential diagnosis and principles of management. Lepr Rev 1975; 46:199-212
33. van Brakel WH, Khawas I. Silent neuropathy: An epidemiological description. Lepr Rev 1994; 65: 350-60
34. Samanta SK, Das S. Recent advances in Ocular Leprosy. Indian J Lepr 2007; 79:135-50
35. Samanta Sk, Sarkar K. Chatterjee PR et al. A current profile of the pattern of ocular leprosy in RFT (released from treatment) era in Eastern India. Abstract: First Asian Leprosy Congress, Agra, India, 2000.
36. WHO Leprosy disabilities: magnitude of the problem. WHO Weekly Epidemiological Record 1995: 70; 269-75.

37. Tiwari VD, Mehta RP. Deformities in leprosy patients of Indian armed forces treated/reviewed at military hospital Agra (a retrospective study). Lepr India 1981; 53:369-78

38. Prevention of disabilities. Lepr Rev 2002; 73 Suppl 1:S35-S43 (Editorial)

39.Job et al –Int J Lepr 1964;32:177-183.

39.Van Brakel WH, Reed NK, reed DS. Grading impairment in leprosy, Leprosy Review 1999; 70:180-88

40.Shah A, Shah N. Prevention and Care of Disabilities in Leprosy Pub; Novartis Comprehensive Leprosy care Association, Mumbai, 2006; 20-24.

41.Kishore J (2000). National Health Programmes of India, Publications, New Delhi, pp132-1332.

42.Central leprosy division –NLEP Monthly progress report for the year 2013 2014.Director General of Health services, New Delhi.

43. District progressive monthly report from district leprosy office Madurai 2013-2014.

44. District progressive monthly report from district leprosy office Madurai 2013-2014.

45.Rea T.H.and Levan N.E.Arch Dermatol 1978:114:1023-1028.

47.WHO guidelines for management of reactions in leprosy.Expert WHO committee on Leprosy World Health Organ Tech Rep.Ser,1998;874:1-43.

48.Ramu G,Desikan KV.Reaction in borderline leprosy.Indian J Lepr 2002; 74;115-28.

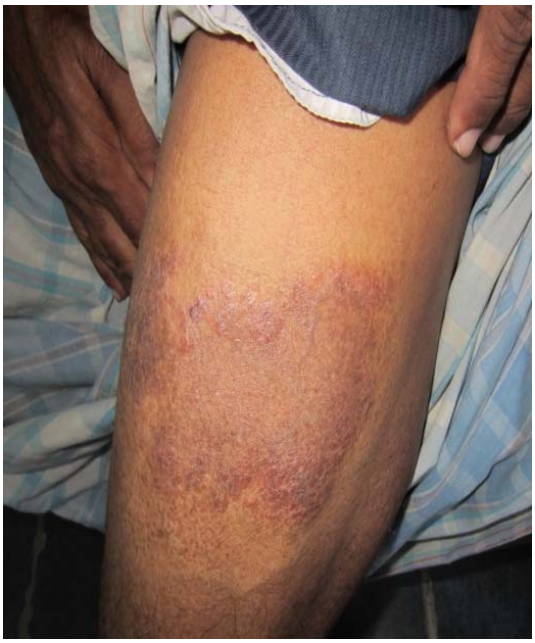
49.Harboe M,Aseffa A ,Leekasa R.Challenges presented by nerve damage in leprosy.Lepr Rev 2005;76:5-13.

50. Park JE. Leprosy. In: Texbook of Preventive and Social Medicine, 16th edn. Park K (Ed), Banarsidas Bhanot (Pub). Jabalpur (India), 2000; 242**.**

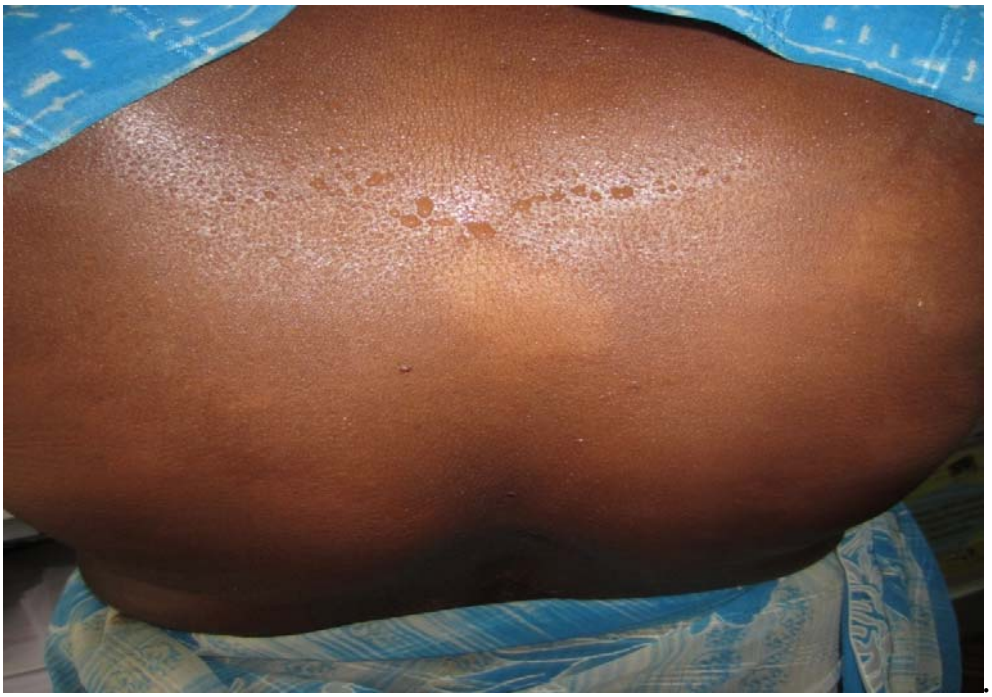
51) Shetty et al Observations from a 'special selective drive' conducted under National Leprosy Elimination Programme in Karjat taluka and Gadchiroli district of Maharashtra. Indian J Lepr 2009, 81 : 189-193

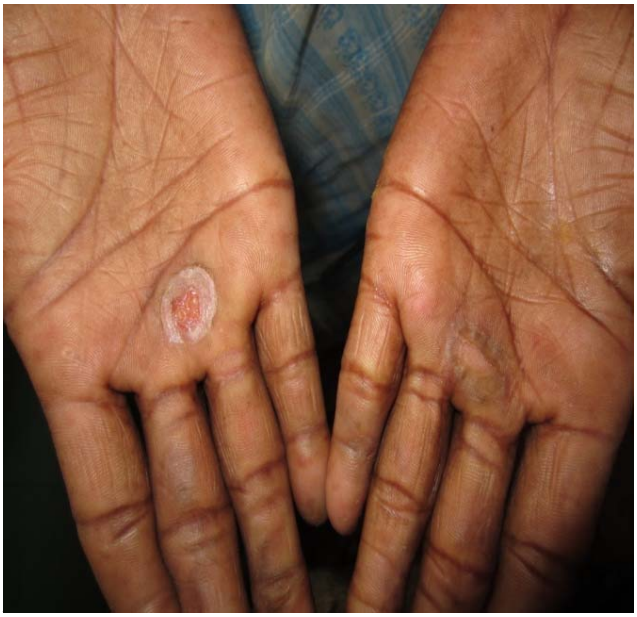
52)Ail kumar et al ‘’The Burden Of New Leprosy Cases in India :A Population – Based survey in Two States.ISRN Tropical Medicine Vol.2013.

BT Hansen with Type 1 reaction

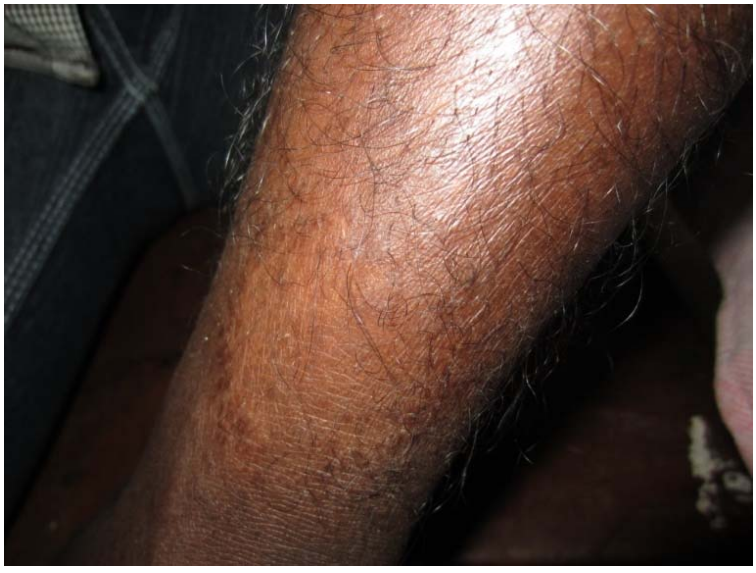


BL Hansen with loss of sweat over the patch and adjacent compensatory hyperhidrosis



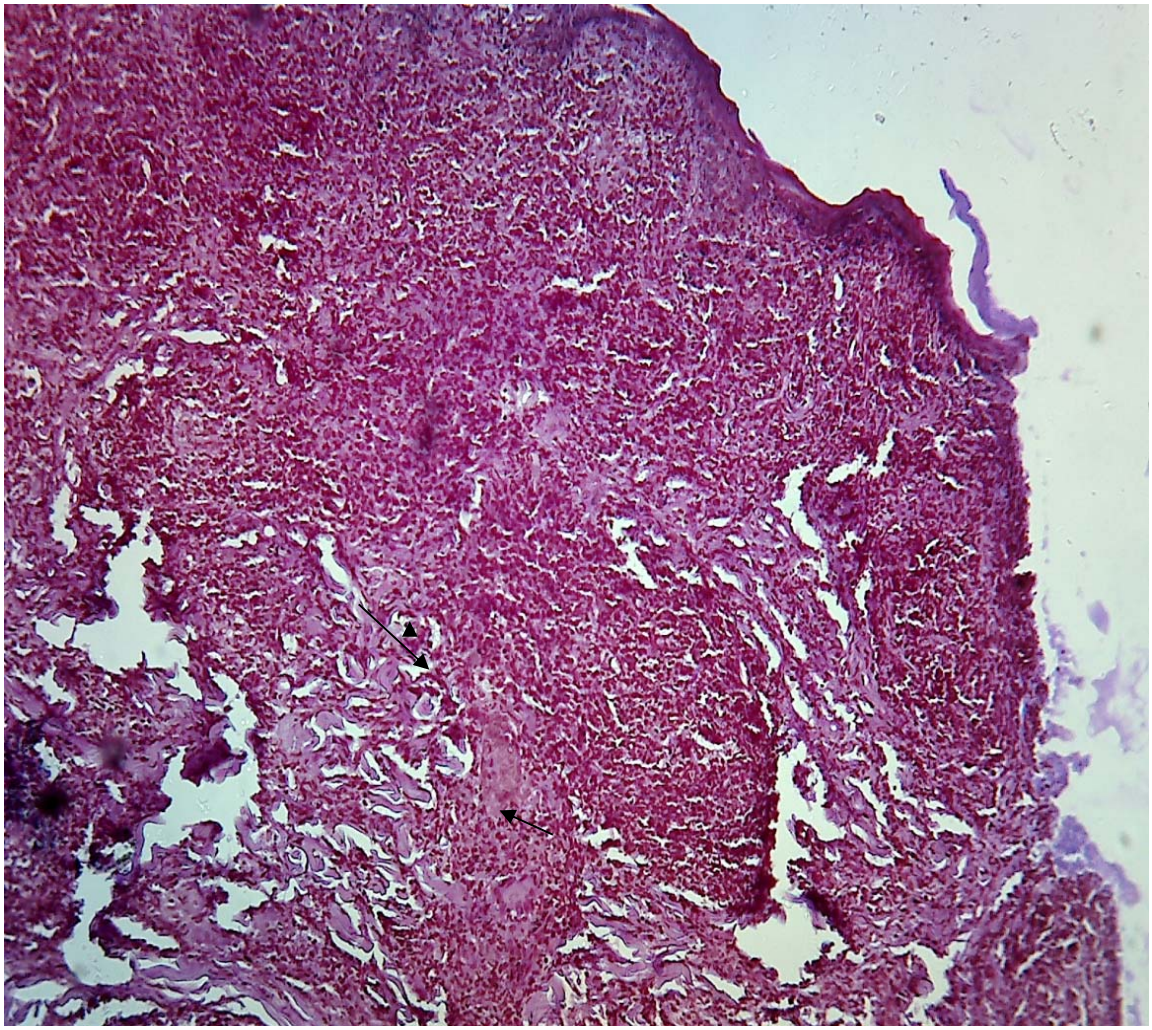


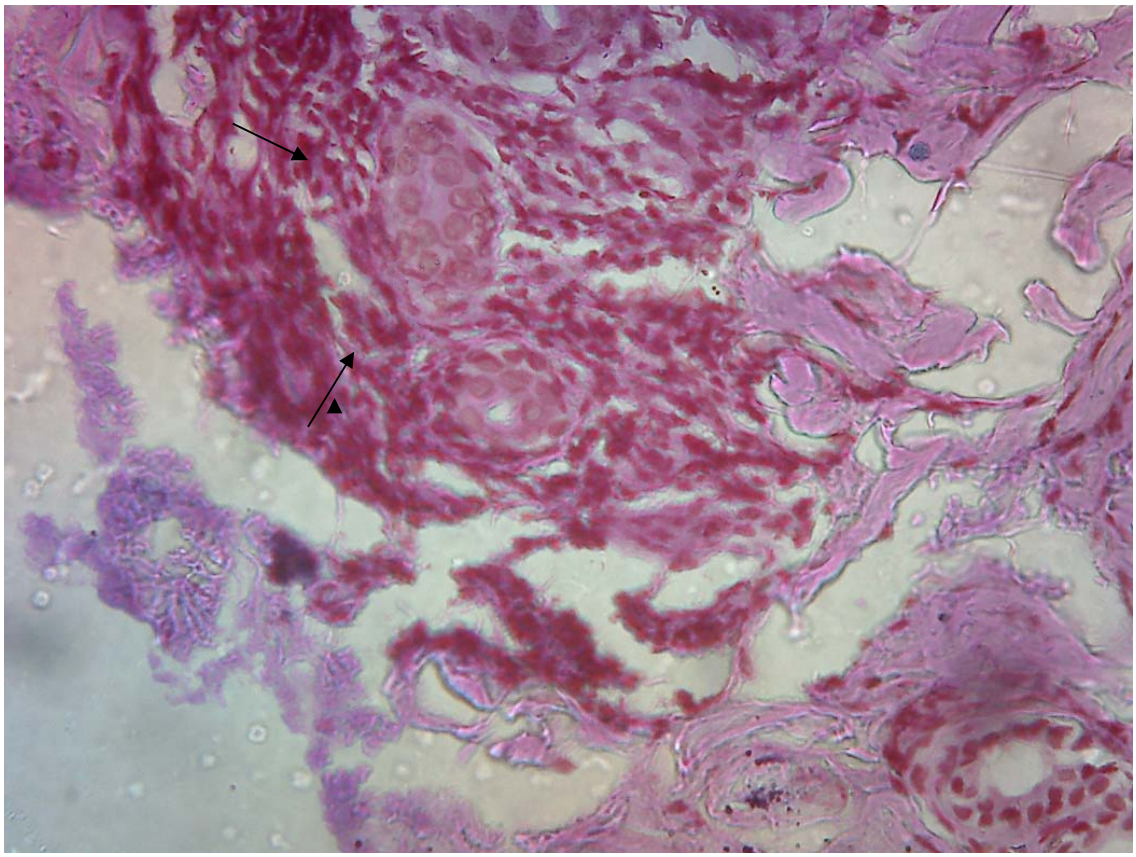
BL Hansen’s with uicer of both palms



Hypo pigmented patch with loss of hair.

Biopsy on low power shows epitheloid granuloma.





Dermis shows epitheloid granuloma with giant cells .

PROFORMA

1. NAME: 2. AGE: 3. SEX: 4.ADDRESS:

5. OCCUPATION: 6. INCOME: 7. SOCIOECONOMIC STATUS:

8. CHIEF COMPLAINTS:

Skin lesion:

Systemic complaints:

9. HOPI:

Onset-sudden/insidious

Progression-slow/rapid

Skin lesion -

Site –

Sensation-loss of sensation/painful.

Constitutional symptoms:fever/athralgia,malaise,fatigue,

Headache.

Neuritis –neural pain/tingling and numbness.

Systemic involment:

Eye-redness/blurring of vision/pain /photophobia

Edema of hands /feet

Swollen and tender testis

Muscle pain/bone pain

Joint pain and swelling

Rhinits /epistaxis.

10. Past history:

DM/HT/other systemic disease.

Hansen in the past-yes/no

If yes - when

Type of Hansen .

(from record /no of patch/no of nerve)

11. Treatment H/O:

ALT taken in the past-

Regimen taken-Dapsone monotherapy/PB-MDT

MBMDT.

Date of Rx started on –

Date of completion-

Default /regular/ dropout-

12. Family history:

Hansen in family members /type of Hansens /treatment taken.

13. Personal H/O:

Married/single

Children

Smoker/Alcohol.

14. General examination: VITALS-P.R: BP:

Febrile SYSTEMS-CVS:

Anemic RS:

Pedal edema ABD:

Generalised lymphadenopathy. CNS:

15. DERMATOLOGICAL EXAMINATION

No and Type of lesions - patch

- papule
- nodule
- plaque
- vesicle /bulla
- ulcer/necrosis.

Margin - well/ill defined

- Extent of margin

Surface - warm

- Shiny /edematous

- Scaly/exfoliation

Colour - erythematous/hypopigmented

Infiltration:

Tenderness:

Site :

Distribution –symmetrical/asymmetrical/ U/L / B/L

Sensation-hypoaesthetic/hyperaesthetic/anesthetic

Other skin lesion - Madrosis

- Nasal depression

- Ear lobe infiltration

- Leonine facies

- Edema of hands/feet

- Gyanecomastia

16. Nerve examination

Cranial nerve: 5^{th} nerve - corneal sensation

- Sensation on face.

7^{th} nerve - facial expression

- wrinkling of forehead

- blinking of eye /Lid lag/deviation of mouth.

17. Peripheral nerves

		Thickening	**Consistency**	**Tenderness**	**Nodularity**
Supra orbital nerve	Right				
	Left				
Facial nerve	R				
	L				
Greater auricular nerve	R				
	L				
Supra clavicular nerve	R				
	L				
Radial nerve	R				
	L				

Ulnar nerve	R				
	L				
Radial cutaneous nerve	R				
	L				
Lateral popliteal nerve	R				
	L				
Post tibial nerve	R				
	L				
Sural nerve	R				
	L				

18. Sensory system :

Anaesthesia along nerve course - partial / complete loss.

Nerve trunk involvement-tem/pain/touch involvement.

Glove and stocking anaesthesia.

19. Acute palsy

Claw hand –partial/total

Wrist drop

Foot drop

Facial palsy

20. VMT - ulnar nerve - little finger abduction card test

Froments sign

Radial nerve –wrist extension

Median nerve –pen test

Lateral popliteal nerve-dorsiflexion of foot

Posterior tibial nerve-eversion of foot

21. Other systems:

Eye –conjunctivitis,iridocyclitis

Arthrits-jt swelling /tenderness

22. Investigations:SSS BIOPSY

23. Diagnosis.

MASTER CHART																												
					Cutaneous						Nerve Thickening			Sensory			Motor Palsy						Skin Biopsy					
S.No	Name	Age	Sex	Place	Hypopigmented/ Erythematous patch	Site	Duration	Single or Multiple	Infiltration	Loss of swet or not	Name of the Nerve	Unilateral	Biletral	Patch	Nerve Distribution	Glove and Stocking	Claw Hand	Wrist drop	Foot drop	Facial palsy	Trophic Ulser	SSS	Epitheloid granulom	Dermal Inflitrate & Foamy macrophage	Diagnosis	New Case	Old Case	Treatement Taken or not
1	Sibiraj	11	M	AGN	HP	KNEE	2m	S						1											BT	PB		
2	Thangaponnu	14	F	AGN	HP	R.FM	1m	S						1									1		TT	PB		
3	Rajesh	22	M	AGN	HP	TRUNK	1-2y	S						1									1		TT	PB		
4	P.Lakshmi	50	F	AGN	HP	F,UL,LL	4y	M	1		LPN,UN		B	1									1		BT	MB		
5	Amudha	40	F	AGN	HP	ELBOW	6m	S	1			U		1									1		BT	PB		
6	Papathy	50	F	AGN	HP	BACK,TRUNK	3-4y	M	1	1	LPN,UN		B								1			1	BL	MB		
7	Sonimuthu	50	M	AGN	HP	TRUNK,LEGS	3m	M	1	1	UN	U					1				1				BT	MB		
8	Sahadevan	49	M	AGN	HP	R.LEG	2-3y	S	1		LPN	U		1									1		BT	PB		
9	Chandran	53	M	AGN	HP	TRUNK	2m	M		1	UN		B												BT	MB		
10	Thothan	50	M	AGN	HP	R.ARM	1y	S			UN	U											1		BT	PB		
11	Mookaiya	50	M	AGN	HP	THIGH	2y	M	1														1		BT	MB		
12	Chinnadurai	34	M	AGN	EP	THIGH	1y	S	1					1									1		BT/T1	PB		

MASTER CHART

					Cutaneous Manifestation				Manifestation			Nerve Thickening			Senory Imparment			Motor Palsy						Skin Biopsy					
S.No	Name	Age	Sex	Place	Hypopigmented/ Erythematous patch	Site	Single or Multiple	Infiltration	Epididymitis	Lymphadenitis	Loss of swet or not	Name of the Nerve	Unilateral	Bileteral	Patch	Nerve Distribution	Glove and Stocking	Claw Hand	Wrist drop	Foot drop	Facial palsy	Trophic Ulser	SSS	Epitheloid granuloma	Dermal Infiltrate & Foamy macrophage	Diagnosis	New Case	Old Case	Treatement Taken or not
1	S.Muthia	60	M	AGN																						DEFAULTER			
2	Muthumal	60	F	AGN																						DEFAULTER			
3	Kamaye	60	M	AGN																						DEFAULTER			
4	Alagappan	50	M	AGN																						DEFAULTER			
5	Palaniammal	52	F	KN																						DEFAULTER			
6	Pandiarajan	13	M	AGN																						OLD CASE ON Rx			
7	Chitradevi	30	F	AGN	HP	R.ARM						UN	U													OLD CASE ON Rx			
8	R.Santhanapandi	29	M	AK	HP	LEG																				OLD CASE ON Rx			
9	Raja	17	M	AK	HP	R.THIGH																				OLD CASE ON Rx			
10	Cheti	34	M	AGN	HP	FACE																				OLD CASE ON Rx			
11	Maduraiveeran	30	M	VMP	HP	TRUNK																				OLD CASE ON Rx			
12	Periyakarupan	60	M	AK	HP	FLANK																				RFT			
13	Manikam	70	M	AK	HP	KNEE						LPN	U													RFT			
14	Theivam	50	M	AK																						RFT			
15	N.Bosh	19	M	AGN																						RFT			
16	Chellamani	51	M	AGN																						RFT			
17	Bathrakali	50	F	AGN																						RFT			
18	Nagamal	70	F	AGN																						RFT			
19	RFT	23	M	AGN																						RFT			
20	Balamurughan	30	M	AGN	HP																					RFT			
21	Veeran	60	M	AGN																						RFT			
22	Muthupandi	40	M	AGN								LPN		B												RFT			
23	Palaniammal	55	F	AGN																						RFT			
24	K.Kannan	60	M	AGN																						RFT			
25	P.Karupusamy	47	M	AGN														1								RFT			
26	Alagu	8	M	AK																		1				RFT			
27	Kilnatan	37	M	AK																						RFT			
28	K.Kumuran	43	M	AK																						RFT			
29	Odiyan	50	F	AGN	HP																					RFT			
30	N.Manikandan	56	M	AGN																						RFT			
31	R.Somu	55	M	AGN																						RFT			
32	P.Ramalakshmi	61	F	AGN																						RFT			
33	S.Laskhmi	40	F	AGN																						RFT			
34	V.Pongodi	45	F	AGN																		1				RFT			
35	Neelamegham	75	M	AGN														1								RFT			
36	Velu	65	M	AGN																						RFT			
37	Andibalan	58	M	AGN	HP																					RFT			
38	Jeyaprakash	50	M	AGN																						RFT			
39	Alaghan	65	M	AGN																						RFT			
40	Prabha	45	F																			1				RFT			
41	Thangachamy	65	M	AGN																						RFT			
42	Malaichami	65	M	AGN																						RFT			
43	Sarasu	56	F	AGN																						RFT			
44	Jeyabalan	60	M	AGN																						RFT			
45	Mookan	65	M	AGN	HP																					RFT			
46	D.Palaniammal	70	F	AGN																						RFT			
47	Selvam	65	M	AGN																						RFT			

48	**K.Nallandu**	**63**	**F**	**AGN**																						**RFT**			
49	**M.Seva**	**55**	**F**	**AGN**																		**1**				**RFT**			
50	**Veeramal**	**43**	**F**	**AK**																						**RFT**			
51	**Ramaya**	**50**	**M**	**AGN**															**1**							**RFT**			
52	**R.Palani**	**42**	**M**	**AGN**																						**RFT**			
53	**Pichi**	**55**	**F**	**AGN**																						**RFT**			
54	**A.Andiappan**	**50**	**M**	**AGN**																						**RFT**			
55	**A.Alaghappan**	**50**	**M**	**AGN**																						**RFT**			
56	**Andiappan**	**50**	**M**	**AGN**																						**RFT**			
57	**Karupannan**	**65**	**M**	**AGN**																						**RFT**			
58	**Nallian**	**65**	**M**	**AGN**																						**RFT**			
59	**Veeran**	**55**	**M**	**AGN**																						**RFT**			
60	**Malaichami**	**50**	**M**	**AGN**																						**RFT**			
61	**Palaniandi**	**55**	**M**	**AGN**	**HP**																					**RFT**			
62	**Alagappan**	**50**	**M**	**AGN**	**HP**																	**1**				**RFT**			
63	**Chinnan**	**52**	**M**	**AGN**																						**RFT**			
64	Andiappan	55	M	AGN																						RFT			
65	**Kasipathi**	**45**	**M**	**AGN**																						**RFT**			
66	**Veeraie**	**65**	**F**	**AGN**																						**RFT**			
67	**Krishnan**	**36**	**M**	**KN**																						**RFT**			
68	**Samayan**	**60**	**M**	**KN**																						**RFT**			
69	**Duraisami**	**50**	**M**	**KN**																						**RFT**			
70	Rathinam	76	M	KN																						RFT			
71	**RAJA**	**50**	**M**	**AGN**																						**RFT**			

KEY FOR MASTER CHART

SEX

M – Male

F – Female

PLACE

AGN – Alanganallur

CUTANEOUS LESIONS

HP – Hypopigmented Patch

EP – Erythematous Patch

S – Single

M - Multiple

SITE

R – Right

L - Left

FM – Forearm

UL – Upper Limb

LL – Lower Limb

UB – Upper Back

DURATION

m – Month

y – Year

INFILTRATION

1 – Present

LOSS OF SWEAT OR NOT

1 – Present

NERVE THICKENING

LPN – Lateral Popliteal Nerve

UN – Ulnar Nerve

U – Unilateral

B – Bilateral

SENSORY IMPAIRMENT , MOTOR PALSY, SKIN BIOPSY, TROPHIC ULCER

1 – Finding Present

DIAGNOSIS

TT – Tuberculoid

BL – Borderline Lepromatous

BT – Borderline Tuberculoid

T1 – Type 1 Reaction

Old Case – On Treatment

RFT – Released From Treatment

NEW CASE

PB – Paucibacillary

MB - Multibacillary

Ref. No. 20735/E4/2/2013 **Govt. Rajaji Hospital,**
Madurai.20. Dated:20.12.2013

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for November 2013
Approved list -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 18.11.2013, Monday at 10.00 am to 12.00.noon at the Anaethesia Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Surgical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. I. Jeyaraj, M.S., (Anatomy) Cell.No 9566211947	Director & Professor Institute of Anatomy /V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048	Professor of Medicine Madurai Medical College	Member
6. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031	Professor & H.O.D of Surgery i/c Madurai Medical College	Member
7. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
8. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
9. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Project was approved by the committee

..2..

Name of P.G.	Course	Name of the Project	Remarks
Dr. V. Kavitha	PG in MD., DVL, Madurai Medical College and Government Rajaji Hospital, Madurai-20.	A Population based survey to assess the current scenario of leprosy in Alanganallur block, South Tamilnadu.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**
Ethical Committee

DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

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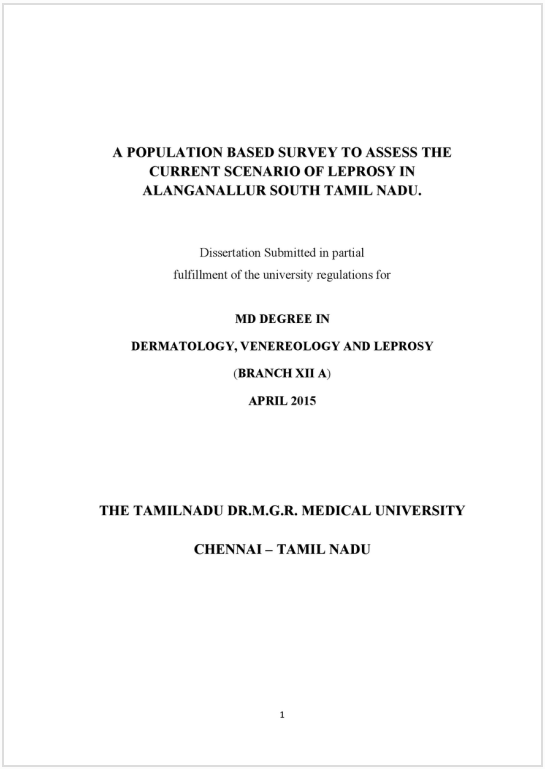

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Dissertation Submitted in partial
fulfillment of the university regulations for

MD DEGREE IN

DERMATOLOGY, VENEREOLOGY AND LEPROSY

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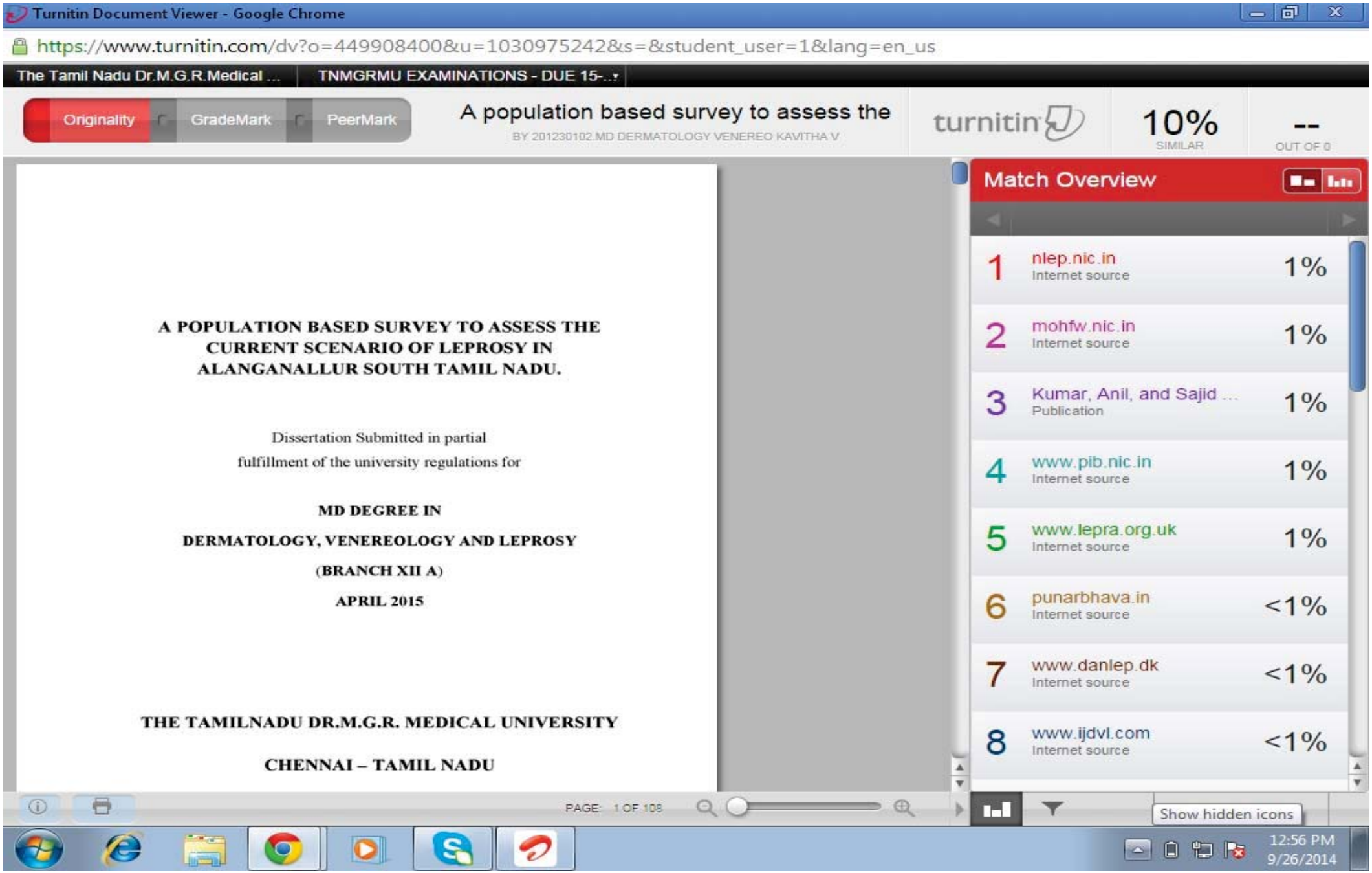
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