"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMATOSCOPY IN LEPROSY AND ITS ASSOCIATION WITH CLINICAL SPECTRUM AND HISTOPATHOLOGY AT A TERTIARY CARE CENTRE."

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JAWAHARLAL NEHRU MEDICAL COLLEGE, KAHER,
BELAGAVI – 590010
KARNATAKA.

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BRDoM.

Dr. BHAVANA R. DOSHI MD, DVD, FIDD
Professor and Head,
Department of Dermatology,
Venereology and Leprosy
J. N. Medical College,
Nehru Nagar, Belagavi – 590010
Date : 02/01/2023
Place : Belagavi

Prof. & Head
Dept. of Dermatology, Venereology & Leprosy
J. N. Medical College, BELAGAVI

KAHER
J.N. MEDICAL COLLEGE
BELAGAVI

DR.(Mrs.) N.S. MAHANTSHETTI MD
Principal
J. N. Medical College,
Nehru Nagar, Belagavi – 590010
Date : 02/01/2023
Place : Belagavi

PRINCIPAL
J.N. Medical College,
BELAGAVI-590 010

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 22-12-2022.

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BT0120002,
Postgraduate Student,
2020-21 Batch,
Department of Skin & V.D,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 27 **Date: 25/01/2021**

To,

REG NO: BT0120002
PG student in Dermatology, Venereology and Leprosy,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMATOSCOPY IN LEPROSY AND ITS ASSOCIATION WITH CLINICAL SPECTRUM AND HISTOPATHOLOGY AT A TERTIARY CARE CENTRE",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

LIST OF ABBREVIATIONS USED

Sl. No.	Abbreviation	Expansion
1	AFB	Acid Fast Bacilli
2	BB	Mid Borderline / Borderline borderline
3	BL	Borderline Lepromatous
4	BT	Borderline Tuberculoid
5	I	Indeterminate
6	LL	Lepromatous
7	PGL	Phenolic Glycolipid
8	SC	Schwann Cell
9	TT	Tuberculoid
10	WHO	World Health Organization
11	MB	Multibacillary
12	PB	Paucibacillary
13	SSS	Slit Skin Smear
14	M. leprae	Mycobacterium leprae
15	M. lepromatosis	Mycobacterium lepromatosis
16	DNA	Deoxyribonucleic acid
17	H & E	Hematoxylin and Eosin
18	MDT	Multi Drug Therapy
19	HIV	Human Immunodeficiency Virus

20	**T1R**	**Type I lepra reaction**
21	**T2R**	**Type II lepra reaction**
22	**PCR**	**Polymerase Chain Reaction**
23	**NTM**	**Nontuberculous mycobacteria**
24	**SES**	**Socioeconomic status**

ABSTRACT

Background: Leprosy is a persistent granulomatous infection affecting mainly the skin and nerves that can manifest clinically in many different ways which makes it a diagnostic challenge and can be easily confused with other infective and non-infective granulomatous disorders.

Dermatoscopy is a useful non-invasive modality which reveals structures and features invisible to the naked eye, and hence can aid in the diagnosis of leprosy. Dermoscopic findings must be associated with clinical spectrum and histopathology as only the combination of such data may significantly improve the diagnostic precision in the field of leprosy diagnosis.

The current study aims to evaluate the dermatoscopic characteristics of several leprosy spectrums and to link the results to clinical spectrum and histopathological findings in the patients.

Materials and Methods: The current cross-sectional study was conducted among 40 treatment naïve leprosy patients who visited the dermatology, venereology, and leprosy outpatient department at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi over a period of one year. After obtaining consent, all participants underwent a detailed history taking, general physical, systemic and dermatological examination. Dermoscopic examination of the most characteristic lesion was done using DermLite DL4 dermoscope with findings being noted along with recording of both clinical and dermoscopic images. Skin biopsy was taken from the same site where dermoscopy was done taking all aseptic precautions and sent for

histopathological analysis. All the data was collected in proforma and analysed statistically.

Results: In the present study, a total of 40 patients fulfilling inclusion criteria were included with a mean age of 43.43 ± 10.09 years of age. Among them, 67.5% were male patients and 32.5% were female patients.

After clinical examination and histopathological analysis, amongst the 40 patients, 14 (35%) cases were of borderline tuberculoid leprosy, 13 (32.5%) cases were of borderline lepromatous leprosy, 5 (12.5%) cases were of tuberculoid leprosy, 5 (12.5%) cases were of lepromatous leprosy and 3 (7.5%) cases were of histoid leprosy.

The dermatoscopic features seen were yellowish orange structureless areas corresponding to dermal granuloma which were seen in all spectrums of leprosy. Broken pigment network was seen corresponding to melanocyte damage. In addition, tuberculoid spectrum showed reduced white dots corresponding to loss of hair follicles and sweat glands due to destruction by peri appendageal granuloma. Lepromatous spectrum showed white chrysalis like structures and white scaling. Histoid leprosy showed blanchable dome shaped structures with peripheral brown pigmentation corresponding to granuloma with spindle shaped cells.

The data was analysed using IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp and Microsoft Excel. The sensitivity of dermoscopic examination with respect to histopathological examination in identifying BT, BL, TT, LL and histoid leprosy were 100%, 76.92%, 100 %, 80% and 100% respectively, and specificity was 88.46 %, 100%, 100%, 100% and 97.3% respectively. Significant

association ($p < 0.001$) was found between the dermatoscopic features and the clinical and histopathological findings by using Fisher's Exact test.

Conclusion: The present study documented the dermatoscopic features of different spectrum of the leprosy. The study also documented significant association of the dermatoscopic findings with the clinical and histopathological findings in the patients. The study concludes that the dermatoscopy is reliable and can be useful in characterizing the spectrum of leprosy with acceptable accuracy at setting with less resources.

Limitation: The limitation in our study was the limited sample size of the study. However, in view of the decreased incidence of leprosy in Indian population and resulting difficulty in procuring treatment naïve cases of leprosy for the study, the small sample size of our study can be considered reasonable.

Key words: Leprosy, Dermatoscopy, Histopathology

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INTRODUCTION

Leprosy is a chronic infectious disease of granulomatous nature with a myriad of clinical presentation which makes it a diagnostic challenge and can be easily confused with other infective and non-infective granulomatous disorders. The utmost comprehensive classification of the disease types is the Ridley Jopling classification which includes Tuberculoid (TT), Borderline Tuberculoid (BT), Mid-Borderline (BB), Borderline Lepromatous (BL), and Lepromatous (LL), and is based on several criteria including clinical, pathological, bacilloscopic, and immunological features.[1]

Although leprosy is a routine diagnosis and a well-known issue, recognition may become difficult where leprosy may be misdiagnosed as many different inflammatory dermatoses.[2]

Dermoscopy is a useful non-intrusive modality which reveals structures and features invisible to the naked eye, providing additional morphologic information during clinical examination of skin lesions and hence can aid in the diagnosis of leprosy.[3]

Dermoscopy has recently emerged as a useful non-invasive method to aid in the identification of granulomatous disorders. Additionally, dermoscopy can help determine the leprosy pole, which is primarily a clinical diagnosis supported by histology. Dermoscopy characteristic patterns will undoubtedly make a rapid and accurate leprosy diagnosis possible for patients.[4]

The clinical spectrum and histology must be linked to dermoscopic findings because only the combination of such data may significantly improve the diagnostic accuracy in the field of detecting leprosy.[4]

Dermatoscopy in diagnosing leprosy is a very novel concept and there is a scarcity of literature associating clinical, dermatoscopy and histopathological findings in leprosy. Determining the value of dermatoscopy in leprosy and its relationship to the clinical spectrum and histopathology at a tertiary care facility was the goal of the current study.

AIMS AND OBJECTIVES

- **PRIMARY OBJECTIVE**

To describe the dermatoscopic features of different spectrum of leprosy.

- **SECONDARY OBJECTIVE**

To associate the dermatoscopic findings with the clinical spectrum & the histopathological findings.

REVIEW OF LITERATURE

INTRODUCTION

Leprosy or Hansen's disease is a chronic granulomatous infection caused by Mycobacterium leprae which mainly affects the integument, the nervous system, mucosal surfaces of the upper respiratory system and the eye. It can appear clinically in a variety of ways, ranging from paucibacillary tuberculoid to multibacillary lepromatous.[1]

Hansen's disease is another name for leprosy. Mycobacterium leprae and Mycobacterium lepromatosis, which both infect the skin and peripheral nerves, are the main causes of this persistent granulomatous illness.[5]

M. leprae and M. lepromatosis are both members of the Mycobacterium leprae complex. Despite the fact that both mycobacteria are classified as separate species according to their DNA sequences, they are both obligate intracellular entities with numerous similarities that induce the same clinical illness.[6,7]

The medical community is very concerned about leprosy. Contrary to popular assumption, this condition is not very infectious, and treatment is easily available. Significant reduction in producing ocular deformities and handicap of the extremities is achievable with increased awareness and early medical care. Relapses are uncommon, but any impairment caused by neuropathy is permanent and may need lifetime support.[8,9,10]

HISTORY

Leprosy is a chronic illness that dates back thousands of years. A Papyrus document from Egypt in around 1550 BC has the oldest record of an illness that many researchers consider to be leprosy. Leprosy like illness is described in Indian texts. Around 1400 BC, it was also mentioned as Kusht in Vedic texts.[11]

In the fourth century BC, soldiers and camp followers returning from Alexander the Great's Greek conquering campaigns in Asia most likely brought the disease from India to Europe. However, Arateus, a Greek physician, first identified the illness as leprosy in about 150 AD and described it as elephantiasis.[12]

Leprosy has been feared since times immemorial, which has led to tremendous stigma and exclusion of individuals who suffer from it. It was believed to be an inherited illness, a curse, and a retribution from the Gods. Leprosy sufferers were required to wear specific attire and ring bells to alert others to their approach throughout the Middle Ages.[13]

Between the 12th and 15th centuries, noble families constructed the Leprosoria , hospitals for people with leprosy. During that time, leprosy victims were regarded as legally dead.[14]

According to Moller Christensen's research, the pathognomonic bone alterations were present in 80% of the skeleton found at Naestved, Denmark.[15]

Norwegian leprosy experts Carl William Boeck (1808–75) and Daniel Cornelius Danielssen (1818–94) held the view that leprosy was a congenital illness rather than an infectious one.[16]

Danielssen's son-in-law, Dr. Gerhard Henrik Armauer Hansen (1841–1912), was the first to discover the leprosy-causing germ under a microscope (1873). Mycobacterium leprae, discovered by Hansen, demonstrated that leprosy was brought on by a germ rather than being inherited, cursed, or the result of sin.[17]

Paul Ehrlich first described the characteristic of acid fastness in 1882. In 1898, Schaffer conducted research on the spread of leprosy through aerosols.[19]

Paul Unna proposed the idea of bacilli grouping into "globi" in 1909 and demonstrated the sub epidermal zone which is cell free in histological sections. The Gillis W. Long Hansen's Disease Centre was founded by the U.S. Public Health Service in 1921 at Carville, Louisiana, also known as "Carville." It developed become a hub for leprosy research and testing as well as a facility for patients to get live-in care.[20]

ETIOLOGY

A member of the Mycobacterium leprae complex, which also comprises Mycobacterium lepromatosis, Mycobacterium leprae, is an acid fast, gram positive bacillus. The generation time of M.leprae is thought to be between 12 and 13 days, and it multiplies more slowly than the second. Less than half of the functional genes for tuberculosis are present in this obligate intracellular bacterium, which is also incapable of being cultured in artificial medium.[8]

M. leprae grows best at temperatures ranging from 27 to 33 degrees Celsius, according to laboratory testing. This backs up the hypothesis that M. leprae tends to spread more quickly in colder regions of the body. These include the skin's surface nerves, upper respiratory tract membranes, and the skin itself. [9] This strain also does

well in nine-banded armadillos, which are often found in the south-central United States and have a core temperature of 34 degrees Celsius. M. leprae has also been identified in chimpanzees, mangabey monkeys, and cynomolgus macaques in addition to armadillos.[21,22]

Since the identification of the M. leprae and M. lepromatosis genomes, it has become clear that both strains' genetic makeup comprises numerous pseudogenes. Additionally, some genes that could act as essential enzymes in metabolic processes are lacking.[21]

A robust cellular response takes place in TT to keep the illness to the clearly defined skin patches or nerves. Infiltrating CD4+ T lymphocytes create well-defined granulomas surrounding dermal nerves that contain epithelioid and multinucleate giant cells. Skin test reactivity or in vitro lymphocyte responses to M. leprae antigens are used to confirm cellular immunity. Reversal responses and erythema nodosum leprosum are caused by spontaneous changes in the immune response.[20,21]

In lepromatous leprosy, there is a lack of M. leprae-specific cellular immunity, which leads to unchecked bacilli multiplication and widespread penetration of the skin and nerves. Histologically, the dermis lacks structured granulomas but is populated by foamy macrophages and a dispersion of CD4+ and CD8+ cells.[19,21] Borderline leprosy is characterized by a progressive decline in cellular responses, which is connected to a higher bacillary load and more frequent cutaneous and neurological involvement.[21]

EPIDEMIOLOGY

Leprosy is frequently observed in underdeveloped nations, while its frequency varies. In 2009, roughly 16 nations reported 1000 brand new cases. The majority of instances were found to be present in India, Bangladesh, Brazil, Indonesia and Nigeria. Leprosy database reveals that not all instances are documented.[8,23]

Leprosy is endemic in various Indian states and union territories, with an annual case detection rate of 4.56 cases per 10,000 people. The frequency of leprosy in the nation is 0.4 per 10,000 people. In 2020-2021, 58.1% of new cases were multibacillary, 39% were women, 5.8% were children under the age of 14, and 2.41% had obvious abnormalities. The prevalence of noticeable abnormalities was 1.1 per million people.[23]

In March 2021, 79,898 leprosy patients were receiving free MDT therapy across the country. Despite the interruption of health services caused by COVID-19 from 2020 to 2021, 65,147 new cases of leprosy were found, diagnosed, and treated for free. The continuity of these critical healthcare services during the pandemic response guarantees that leprosy is treated and eradicated.[23]

PATHOPHYSIOLOGY

Transmission of leprosy is not fully known; however, it is thought to disseminate via respiratory routes. Individuals with untreated lepromatous infections typically have a high bacilli count.[24,25] Once within the body, the main mechanism of diffusion begins in the upper part of the respiratory system. Reports state that skin injury may also allow host infection to spread. M. leprae causes demyelination of

nerves and axonal conductance loss because it predominantly targets Schwann cells (SCs), which clinically appears as loss of sensation.[26]

The fundamental means for M. leprae transmission and the parameters of the environment that support the bacterium are currently being investigated. M. leprae has demonstrated pliability and survival following amoeba ingestion.[26–28]

Through the transmission of the bacterium to their eggs, ticks have also been observed to assist in transmission. To determine viability, further research can be done to pinpoint reservoirs and vectors using contemporary technical developments in molecular identification. It is vital to remember that many people do not get the disease after being exposed to it. Many variables influence the disease's growth and progression, including immunological status and genetics.[29]

Responses of the Th1 immune system are robust and are related with lower bacterial numbers and limited illness. On the other hand, Th2 responses are feeble and are associated with raised bacterial numbers and grave illness.[29]

Portal of entry

The two main entry routes are through the respiratory route and skin erosions. Bacilli can also enter the body through the gastrointestinal system and by transplacental transfer.[30]

Mode of transmission [31,32]

The primary mechanism of transmission is inhalation (droplet infection).

Other methods include close physical skin-to-skin contact, in utero transmission, breast milk, post trauma, and insect-borne transmission.

PREDISPOSING CAUSES

Leprosy transmission is caused by a number of reasons, including;

Close contact: Compared to the general public, having close proximity with a leprosy patient significantly increases the risk of acquiring leprosy.[29]

Armadillo exposure: The nine-banded armadillo is the natural host of the M. leprae strain in the southern United States. Although the mechanism of transmission of bacterium from armadillos to people is poorly understood, molecular typing tests have demonstrated the zoonotic transmission.[33]

Age: Elderly people are more vulnerable to contracting leprosy. According to certain research, age has a bimodal connection. Elevated risk was detected between 5 and 15 years of age, with sustained risk beyond 30.[34]

Genetic Influences: As previously stated, genetics influences the immune response. Genetic factors, especially the PARK2/PACRG gene, are thought to be responsible for innate immunity. A study including over 1000 individuals with new leprosy diagnoses and twenty one thousand contacts revealed that genetic relationships were crucial. These associations demonstrated the importance of genetics as a risk factor, regardless of distance between contacts.[34,35]

Immunosuppression: This infection is more likely to occur once the immune system has been suppressed. Leprosy usually develops following a solid organ transplant, chemotherapy, HIV infection, or the use of rheumatologic drugs.[36]

CLASSIFICATION [37,38]

DANNIELSSEN AND BOECK (1848) - 1. Nodular 2. Anaesthetic

NEISSER (1903) - 1. Lepra tuberosa 2. Lepra cutanea 3. Lepra nervorum

PAN AMERICAN (1946)- 1. Tuberculoid 2. Lepromatous 3. Un characteristic

MADRID (1953) - 1. Lepromatous type(L) : Macular, Diffuse , Infiltrated , Nodular , Pure neuritic ; 2 . Tuberculoid type(T) : Macular(Tm) , Minor tuberculoid(Tt) , Major tuberculoid(TT), Pure neuritic (Tn); 3. Indeterminate group(I) : Macular (Im) , Pure neuritic (In) ; 4. Borderline group(B) : Infiltrated , others.

REVISED INDIAN CLASSIFICATION (1981): 1. Tuberculoid 2. Borderline 3. Lepromatous 4. Indeterminate 5. Pure neuritic

RIDLEY AND JOPLING (1962) [39]:

This is most widely accepted classification used currently.

It is based on clinical, bacteriological, histological and immunological parameters and is classified into 5 types: 1. Tuberculoid (TT) 2. Borderline tuberculoid (BT) 3. Borderline Borderline (BB) 4. Borderline Lepromatous (BL) 5. Lepromatous (LL)

WHO CLASSIFICATION (1998) : 1. Paucibacillary single lesion leprosy(SLPB) 2. Paucibacillary leprosy (PB) 3. Multibacillary leprosy (MB)

PB : 1– 5 skin lesions , no nerve / only one nerve , skin smear negative at all sites.

MB : 6 and above skin lesions , more than one nerve irrespective of number of skin lesions , positive skin smear at any site.

CLINICAL FEATURES

Leprosy is an entity with several manifestations, owing primarily to the widespread immunological response to the Mycobacterium leprae strain.[37] The “Ridley Jopling classification” encompasses the complete breadth of signs and symptoms. From a strong immunological response involving few tuberculoid organisms to a modest reaction including a large number of multibacillary cells. In general, this categorization is based on the body's cutaneous, biopsy, and neurologic results. This provides medical practitioners with a good idea of the immunological response that will be generated. The aforementioned features are also linked to AFB found in dermis.[38,39]

Tuberculoid leprosy

Large erythematous or hypopigmented lesions with distinct, elevated borders characterize this entity. Presentation of scaly plaques was seen.[44]

Borderline tuberculoid leprosy

It is characterized by presence of macules with a "target" appearance. The number of skin lesions is more than that in TT and typically manifests asymmetrically in this particular condition. These lesions are referred to as "paucibacillary" when using the WHO classification that was previously stated.[44]

Mid borderline leprosy

With its "punched out" lesions, the illness mostly resembles borderline tuberculoid leprosy or borderline lepromatous leprosy. Most of the central parts are anesthetic.[44]

Borderline lepromatous leprosy

Erythematous macules, nodules, or papules are examples of lesions in this situation; they don't have a clear pattern of occurrence on the body. Although it is possible to find normal patches of skin, the lesions are difficult to distinguish and are widely dispersed. It has been demonstrated that larger lesions are distributed disproportionately.[44]

Lepromatous leprosy

Significantly advanced cases exhibit earlobe nodular growths combined with body hair loss. Mucosal invasion can have nasal stuffiness, comparable to a cold. Without prompt treatment, a collapse or perforated septum may happen. Also, sporadic bacteremia during this illness may appear asymptomatically. In this instance, M. leprae advances with isolated lesions in many organs. Some circumstances reveal the presence of microorganisms in the liver or bone marrow after biopsies have been sought. Larynx and testicles may also be involved.[45,46]

Histoid leprosy

It is considered to be an unusual variant of LL and is characterized by numerous skin colored, dome shaped, shiny, succulent subcutaneous nodules mostly

over the extensor aspects of extremities. The lesions show numerous longer bacilli in clusters with the absence of globi.

Neuropathy

The widespread reduction in sensation in lesions indicates nerve association early in the course of leprosy. Damage to peripheral nerves is a major concern, and reliable analysis of the peripheral nerves is essential for efficient assessment. Although pain can sometimes occur later in the course of the disease, neuropathy usually begins with a loss of sensory perception.[47,48]

Ophthalmic injury

Corneal abrasion and ulceration, lagophthalmos and dryness could all result from decreased innervation of the nerves that control the eyelid muscles and transmit the corneal signals. Thorough examinations require complete ocular assessments.[48]

IMMUNOLOGIC REACTIONS:

These are systemic reactions, and they can happen before the start of treatment, throughout the course of treatment, or even a while after the course of treatment has ended. The two types of lepra reactions are Type 1 reversal reaction (T1R) and Erythema Nodosum Leprosum (T2R) (ENL). Lepromatous disease commonly has T2R, but patients with borderline condition typically have T1R. More research is required for each of these reactions, particularly on the underlying processes and prerequisites for their persistence.[48,49]

Type I lepra reaction (T1R) [48,49]

Typically happens in BT, BB, or BL situations. The presentation encompasses:

- Erythematous, edematous areas with existing lesions associated with nerve trunk
- Erythema of pre-existing skin lesions
- Inflammation leading to deformity and paralysis
- Oedema
- Ulceration
- Decreased or loss of nerve function

Type 2 lepra reaction (T2R) / Erythema Nodosum Leprosum (ENL)[48,49]

Most commonly seen in BL and LL spectrum. Type 2 reactions are characterized by the abrupt emergence of tender nodules in the dermis that may be deep or superficial. Excessive pus discharge might result in the production of pustules. Polymorphs and acid-fast bacilli have been found in the exudate. Lesions are most commonly found on the surfaces of limbs and have a short life expectancy.

The Lucio phenomenon is a very uncommon illness that begins with necrotizing vasculopathy in people with undiagnosed lepromatous leprosy and can occur in situations with Type 2 response.[50]

EVALUATION

Histopathological investigation utilizing skin samples and PCR was important to the development of laboratory procedures. When assessing for leprosy using laboratory testing, the following was shown to be common: raised leukocytes, reduced hemoglobin, poor hematocrit, deranged liver function tests, and raised serum CRP (C reactive protein).[47,50]

SLIT SKIN SMEAR

This is useful for the diagnosis, classifying leprosy into different spectrum, treatment monitoring and for assessing the prognosis of the disease.

SITES: Earlobe is the most common site followed by forehead, chin, dorsal surface of the fingers, extensor surface of the forearms and knees, buttocks and from the skin lesions. At early stage bacilli are demonstrated from nasal smear and pulp of fingers.[51,52]

WHO recommends a minimum of three sites, i.e., one earlobe and two active lesions for smears and in case of a single lesion, the two smears should be taken from diametrically opposite active edges of the lesion. [53]

METHODS: In slit method, after cleaning with spirit, the earlobe is pinched tightly between index finger and thumb for a few minutes to obtain a bloodless field. Using a Bard Parker blade (No. 15), an incision measuring 5 mm in length and 3 mm in width is made, and scraping is then taken. On the glass slide, a smear between 0.7 and 1 centimeter in diameter is created. The Ziehl-Neelsen method is used to stain the slides.

After microscopic examination of slides, the following indices are calculated.

INDICES [51]

A. Bacteriological Index (BI)

This indicates the total number of bacilli both live and dead seen in an average microscopic field after examining at least 25 microscopic fields.

The index is recorded as follows (Ridley's logarithmic scale for Bacteriological Index).

6+ is many clumps of bacilli in an average field (over 1000)

5+ is 100 – 1000 bacilli in an average field

4+ is 10 – 100 bacilli in an average field

3+ is 1 – 10 bacilli in an average field

2+ is 1 – 10 bacilli in 10 fields

1+ is 1 – 10 bacilli in 100 fields

B. Morphological Index (MI)

In this living bacilli are counted after counting 200 bacilli and given as percentage. It is more specific than BI.

Skin biopsy [10,49,50]

A complete biopsy that includes the subcutaneous tissues is suggested from the lesion's most active and dynamic margin due to the extent of the lesions and the invasion of the nerves. Sectioning with haematoxylin and eosin (H & E) demonstrates the previously reported extreme variance in the spectrum.[49,50]

Importance of skin biopsy

- ✓ To confirm the diagnosis.
- ✓ To classify leprosy.
- ✓ To identify the complications like lepra reactions.
- ✓ To help in the management of leprosy.

Site

The center of the lesion, where the lesion is active, should be used for the biopsy in cases of indeterminate leprosy. If there are many lesions, the one that is the most active will be chosen, and a biopsy will be taken from the lesion's edge.

Size

A biopsied skin of size of 1.5 cm long and 0.6 cm wide with the depth of the dermis and subcutis should be taken.

Fixatives

Lowy's fixative (FMA)

Formaldehyde (40%) - 100ml

Mercuric chloride - 20 g

Glacial acetic acid -30 ml

Distilled water -1 litre

The biopsy sample must be left in this solution for two hours before being moved to 70% ethyl alcohol, where it can be preserved for a long time. These stains have been applied:

- S-100 stain
- Fite-Faraco stain
- Gomori methanamine silver stain
- Haematoxylin and eosin stain

The most widely used staining techniques are the Fite-Faraco and H and E stains.

In all instances, acid fast bacilli will be checked in sections stained with Modified Fite's stain. Based on the Ridley and Jopling Scale, findings on histopathology will be categorized as “Polar Tuberculoid”, “Borderline Tuberculoid”, “Mid-Borderline”, “Borderline Lepromatous”, and “Polar Lepromatous”.

Grade	**AFB Stain**
Tuberculoid (TT)	Non visible
Tuberculoid (BT)	0 to 2+
Mid- Borderline (BB)	3+ to 4+
Borderline Lepromatous (BL)	4+ to 5+
Polar Lepromatous (LL)	5+ to 6+

Polymorphonuclear leukocytes are indicative of type 2 reactions, but fibrin thrombi are seen in lesions known as Lucio's phenomenon. Currently, investigations are being conducted to further refine the histological criteria for the diagnosis of type I reactions. It is critical to ascertain that cutaneous granulomatous infections, such as Mycobacterium tuberculosis and Non Tuberculous Mycobacteria (NTM), do not interfere with prospective examination of mycobacterial cultures.[50]

HISTOPATHOLOGY

BORDERLINE TUBERCULOID[54,55]

- ✓ Atrophy of epidermis.
- ✓ Although there may be a clear subepidermal zone present, granuloma spurs can occasionally invade the epidermis.
- ✓ The granulomas are poorly formed and made up of lymphocytes, Langhan's giant cells, and epithelioid cells. The granulomas branch early in the disease and project as spurs along the neurovascular bundle.
- ✓ Granulomatous swelling of the nerves and possible mild lamination of the perineurium due to lymphocyte infiltration are both present.
- ✓ AFB stain: BI is 0 to 2+.

TUBERCULOID LEPROSY [55-58]

- ✓ Epidermis is atrophied
- ✓ Flat rete ridges
- ✓ The Grenz zone, which is normally invaded by inflammatory cell foci, is absent.

- ✓ Dermis exhibits tuberculoid granuloma, which are composed of clusters of epithelioid cells with a some Langhans giant cells and are encircled by a well-defined rim of lymphocytes. Caseation rarely occurs. The papillary layer of the dermis and the deeper dermis may be affected by the granuloma, which atrophies and erodes the epidermis.
- ✓ Additionally, epithelioid granulomas can be found close to vessels and appendages.
- ✓ Nerves in the dermis are extensively destroyed and are encircled by well-formed epithelioid granulomas.
- ✓ Acid fast bacilli absent.

MID BORDERLINE [55]

- ✓ Epidermis is atrophic.
- ✓ Clear subepidermal zone is present.
- ✓ The dermis contains diffuse granulomas, which are composed of macrophages and epithelioid cells in the ratio of about 1:1. In granuloma, lymphocytes are sparsely distributed and few in number.
- ✓ The differentiating feature from BT is the absence of giant cells.
- ✓ Mild to moderate intercellular edema is seen.
- ✓ Granuloma infiltrates nerves but does not totally destroy them. There is reactive perineurium growth and lymphocytic infiltration.
- ✓ AFB: BI is 3+ to 4+.

LEPROMATOUS LEPROSY [55]

- ✓ Epidermis is thin and atrophied.
- ✓ Rete ridges are completely flattened.
- ✓ Clear subepidermal zone is present.
- ✓ Dermis reveals granulomas consisting of macrophages. Most macrophages initially have pink, granular cytoplasm; however, in older lesions, the cytoplasm turns foamy, vacuolated called as "Virchow cells or Lepra cells".
- ✓ The lesion is riddled with a few lymphocytes and focal clumps of plasma cells. Early lesions typically only show a single small focal cluster of cellular infiltrates; however, as the disease progresses, these clusters coalesce to form a band of infiltration that may extend into the subcutaneous fat. Plaques and nodules are produced when macrophages and other inflammatory cells accumulate and raise the skin above them.
- ✓ The macrophages also invade the nerves. Perineurium rarely proliferates in a reactive manner.
- ✓ AFB stain: All structures exhibit bacilli in clumps. BI is 5+ to 6+.

BORDERLINE LEPROMATOUS [57,58]

- ✓ The transparent subepidermal zone (Grenz zone) divides the granuloma from the atrophic epidermis.
- ✓ Poorly developed granulomas, which are primarily composed of macrophages with a single cluster of epithelioid cells, are visible in the

dermis. Plasma cells and lymphocytes can also be detected. There could be foamy alterations in some of the macrophages.

- ✓ Concentric perineural proliferation creates the look of an onion peel. Numerous lymphocytes and macrophages are present in the perineurium.
- ✓ All structures exhibit AFB clumps.
- ✓ AFB stain: Numerous bacilli with small globi are seen. BI is 4+ to 5+.

HISTOID LEPROSY [59-61]

- ✓ Atrophic epidermis.
- ✓ There is clear subepidermal zone.
- ✓ Circumscribed lesion is usually located in deep dermis or subcutis and is surrounded by pseudo capsule.
- ✓ The lesion is expansile in nature and consisting of spindle shaped histiocytes. Bacilli are arranged in parallel bundles aligned along the long axis of the histiocytes (Histoid habitus). These bacilli are longer than the normal lepra bacilli.
- ✓ Presence of foci of epithelioid cells in the lesion is known as tuberculoid contamination.

Polymerase chain reaction

DNA from M. leprae and M. lepromatosis can be easily detected in tissue by using the laboratory technique PCR. When employed as a detector rather than an identifier, PCR is more useful. Current research found that biopsy PCRs had more

than 90% sensitivity and 100% specificity. Results indicated a sensitivity of 34% and a specificity of 80% in instances with tuberculoid illness.[62]

Serology test

The phenolic glycolipid-1 (PGL-1) specific to M. leprae is cited in serology investigations, but it is not commonly used in clinical practice in the United States since it is not highly sensitive without histological and clinical evidence.[63-65] An increased polyclonal immune response to Mycobacterium leprae phenolic glycolipid-1 (PGL-1) is seen in those with lepromatous disease (PGL-1) with a high rate of false-positive outcomes. Because tuberculoid illness does not usually cause the development of PGL1 antibodies, this method of testing is ineffective for this group of individuals.

Differential diagnosis[5,65]

When the patient perceives total sensory loss over the lesion, including mild touch and pin pricks, this is confirmatory evidence of leprosy. Typically, a skin biopsy is used to establish the diagnosis. Differential diagnosis includes:

- Granuloma annulare
- Fungal infection
- Annular psoriasis
- Systemic lupus erythematosus
- Keloid
- Mycosis fungoides
- Neurofibromatosis
- Cutaneous leishmaniasis

The diagnosis of type 1 reactions is generally accomplished only by clinical assessment. Standard lab tests are not easily available to assist in diagnosis. CXCL10 chemokine levels in the blood have been linked to type 1 responses. CXCL10 is not to be used as a T1R indication. Because CXCL10 has not been demonstrated to be present in large quantities prior to the response, there is little confidence in its predictive abilities.[57,58]

DERMATOSCOPY / DERMOSCOPY

A dermoscope or dermatoscope is a diagnostic non-invasive tool which is used to see the structures of skin not visible to the naked eye. It uses the principle of transillumination of a lesion with magnification for visualization of the lesion. Incident light falling over a dry and scaly skin gets reflected whereas over oily skin it reaches till reticular dermis. Hence linkage fluids such as oil (immersion oil, mineral oil), water or glycerin is used. Also contact method is preferred than non-contact method as transillumination is enhanced by the contact of glass over the lesion containing the lesion fluid. This is because refractive index of glass is 1.52 and is similar to that of the skin (1.55). There are three different modes in a dermoscope, i.e., polarized light, non-polarized light and blue light. The deeper structures of the skin are visualized via polarized light whereas superficial structures are seen via non polarized light.[68]

DERMOSCOPY IN LEPROSY

The literature on dermoscopy in leprosy is extremely scarce. Very few studies have been done on the topic and even fewer studies have associated it with clinical spectrum and histopathology.

Different spectra of leprosy have different patterns on dermoscopy. However, yellowish orange structureless areas are seen in all spectra of leprosy corresponding to the presence of dermal granuloma on histopathology.[69] Tuberculoid spectrum shows loss of hair follicles and eccrine structures in the lesional skin along with broken pigment network and peripheral linear branching vessels.[69] Borderline tuberculoid spectrum shows yellow dots and globules corresponding to dilatation of pilosebaceous unit in addition to the features seen in tuberculoid leprosy.[70] Borderline lepromatous leprosy shows white shiny streaks or chrysalis like areas corresponding to perifollicular hyperkeratosis on histopathology along with broken pigment network.[70] Lepromatous pole shows accentuation of the normal reticular pigment network with characteristic white scaling.[71] Histoid leprosy shows central hypopigmented and blanchable dome shaped structures with central keratotic plug and perilesional hyperpigmentation with peripheral crown vessels.[70]

Treated and treatment undergoing cases of leprosy show few variations in dermoscopic patterns. Due to the lack of ongoing inflammation in treated cases of leprosy, yellowish orange hue and vascular structures are usually missing.[69] Also there is an increase in basal pigmentation post treatment in leprosy lesions on histopathology.[72] This too might lead to change in dermoscopic patterns in leprosy.

ROLE OF DERMOSCOPY IN LEPROSY: Dermatoscopy can be a useful tool and an adjunct to histopathology for aiding in the diagnosis as well as classifying leprosy into different spectra based on the different patterns seen via a dermoscope. It can aid in the early diagnosis of leprosy as the features seen on dermoscopy may become evident even before clinical presentation. Also, it may differentiate leprosy patches from other similar diseases like vitiligo by the presence of altered pigment network,

micro scaling, and absence of hypo or depigmented islands and perifollicular depigmentation, thus eliminating the need for SSS or skin biopsy especially in areas where histopathology facilities are unavailable.[73,74] The absence of skin appendages and loss of pigment network in leprosy lesions on dermoscopy helps to differentiate it from other granulomatous diseases .[71,74] This proves dermoscopy to be a simple and effective tool with multifaceted benefits.

MATERIALS AND METHODOLOGY

- Study source: This study was conducted in the Department of Dermatology, Venereology and Leprosy, in tertiary care hospital, Belgaum as a part of the MD academic curriculum.
- Study duration: The study was conducted between 1st January 2021 to 31st December 2021.
- **Ethical clearance:** Clearance was taken from the Ethical Committee of the institute.
- **Study design:** Hospital based cross sectional study.
- **Sample size**: Kappa test was used for sample size calculation by using statistical software R from package kappa Size.

By assuming Kappa value at alternative hypothesis as 0.7 with 5% level of significance and 85% power, minimum sample size required was 25 leprosy subjects.

Minimum sample size required was 25. Larger the sample size, better the precision.

Hence, a total number of 40 patients of treatment naïve leprosy were taken into the study.

- **Sample selection criteria:**

  All patients between the age group of 18-65 years of clinically diagnosed treatment naive cases of leprosy attending KLE's Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi were recruited.

- **Inclusion criteria:**

  Patients between the age group of 18-65 years of clinically diagnosed treatment naive cases of leprosy.

- **Exclusion criteria:**
  - Non consenting patients.
  - Lepra reactions
  - Pure neuritic leprosy
  - Patients undergoing treatment/ completed treatment for leprosy
- **Data collection:**
  - Informed consent was taken from all the study patients.
  - All the study subjects were made to go through a detailed history taking, general physical, systemic and dermatological examination.
  - Patients were classified into different spectrum of leprosy according to Ridley Jopling classification. Few cases of histoid leprosy were additionally included in the study.
  - Data was collected by a single examiner and recorded in case record proforma.
  - All participants underwent dermoscopic examination of the most characteristic lesion using DermLite DL4 handheld dermoscope , using contact polarised mode at 10x magnification and images were captured by a OnePlus phone with findings being noted along with recording of both clinical and dermoscopic images.

- Skin biopsy was taken from the same site where dermoscopy was done taking all aseptic precautions and sent for histopathological examination (using Haematoxylin and Eosin staining)
- Records were maintained and analysed statistically.

- **Statistical Method for Data Analysis**

- All the data was collected in proforma and entered in excel sheet.
- The collected data demographic data was summarized as frequency, percentage, mean and standard deviation.
- Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.
- Results on continuous measurement was presented as Mean and SD and categorical as frequency and percentage.
- Inferential statistics like Fisher Exact test was applied.
- P value <0.05 was considered statistically significant.
- Criterion validity in terms of sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) with confidence intervals (CI 95%) were computed using a two-way table with histopathological diagnosis as a gold standard test.

RESULTS

In our study 40 patients who completed the study were included. The mean age group of patients was 43.43±10.09 years. At final analysis there were 27 males and 13 females with male to female ratio of 2.07 :1. Minimum and maximum age group of participants in the study was 19 and 62 years respectively. Minimum and maximum duration of symptoms was 6 months and 24 months respectively with a mean of 14.83±6.41 months.

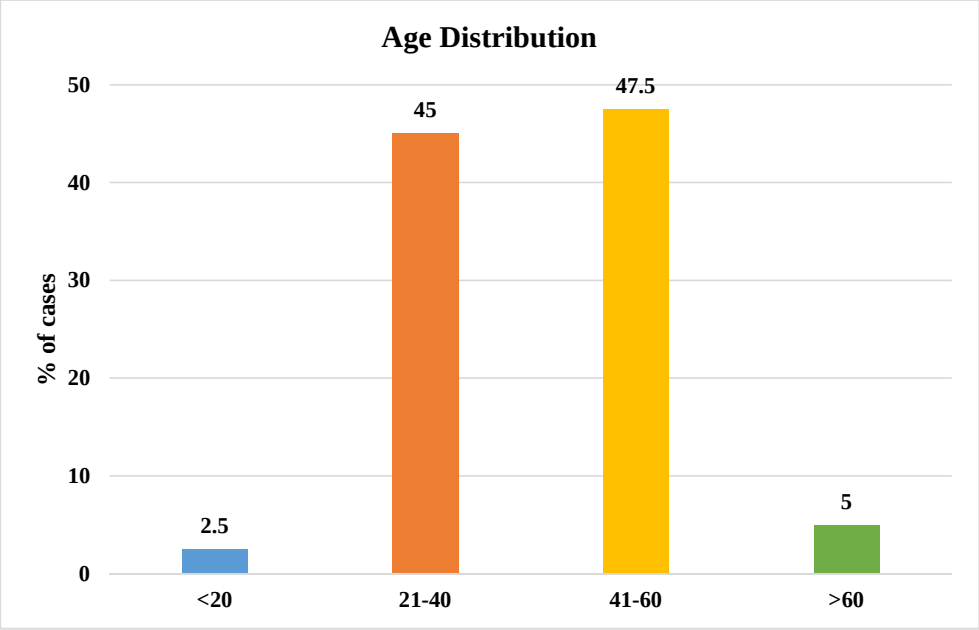
1. Age distribution

Out of 40 patients of leprosy, majority of them i.e., 47.5% (n=19) were in the age group of 41-60 years. This was followed by 45% (n=18) in the age group of 21-40 years. 5% (n=2) patients aged more than 60 years while 2.5% (n=1) were below 20 years. The mean age of the patients was 43.43±10.09 years.

Table 1: Distribution of patients by age (years)

Age (Years)	No. of Cases (N)	Percentage (%)
<20	01	2.5
21-40	18	45.0
41-60	19	47.5
>60	02	5.0
Total	40	100.0
Mean ± SD (Range)	43.43±10.09 (19-62 years)	

Graph 1: Distribution of patients by age (years)



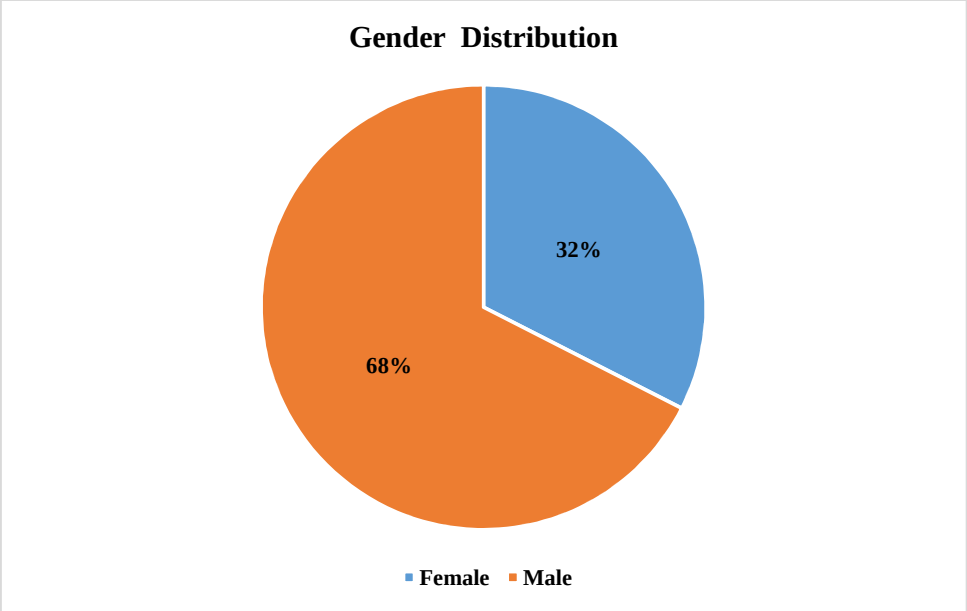
2. Gender distribution:

There was a male preponderance seen, i.e., 67.5% (n=27) of the study population were males and the remaining 32.5% (n=13) were females.

Table 2: Distribution of patients by gender

Gender	No. of Cases (N)	Percentage (%)
Female	13	32.5
Male	27	67.5
Total	40	100.0

Graph 2: Distribution of patients by gender



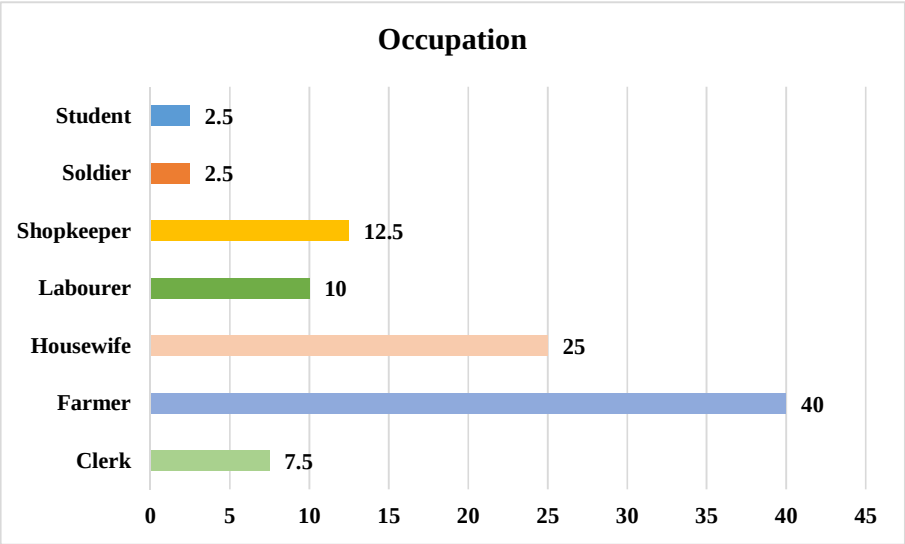
3. Occupation

Maximum number of patients, i.e. 40% (n=16) were farmers by occupation.

Table 3: Distribution of patients by occupation

Occupation	No. of Cases (N)	Percentage (%)
Clerk	03	7.5
Farmer	16	40.0
Housewife	10	25.0
Labourer	04	10.0
Shopkeeper	05	12.5
Soldier	01	2.5
Student	01	2.5
Total	40	100.0

Graph 3: Distribution of patients by occupation



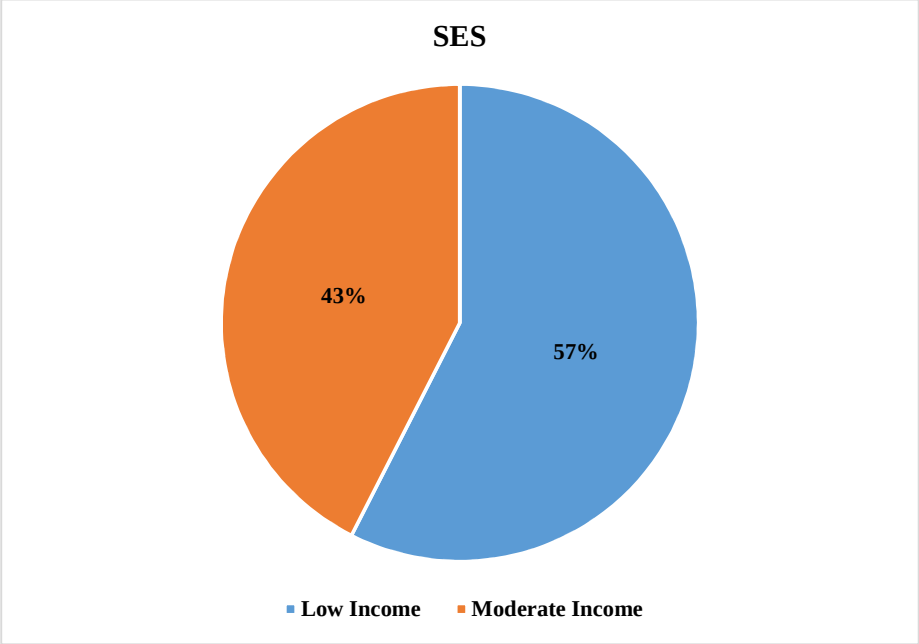
4. Socioeconomic status (SES)

In our study, 57.5% patients were from low-income group whereas 42.5% were from moderate income group. No patients were from high income group.

Table 4: Distribution of patients by SES (Socioeconomic status)

SES	No. of Cases (N)	Percentage (%)
Low Income	23	57.5
Moderate Income	17	42.5
Total	40	100.0

Graph 4: Distribution of patients by SES (Socioeconomic status)



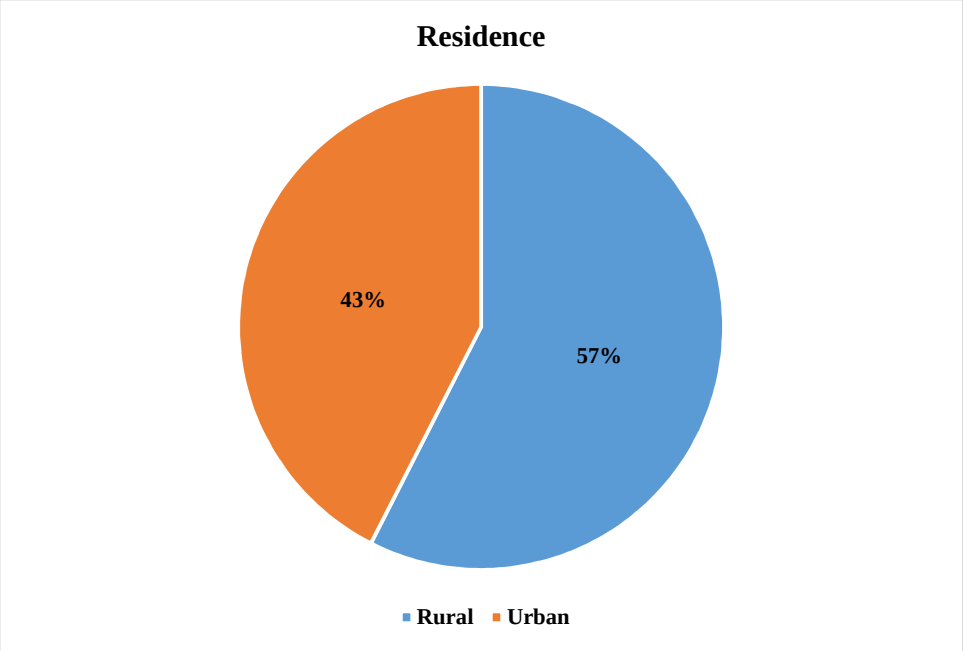
5. Residence

In our study, 57.5% patients hailed from rural areas whereas 42.5% patients hailed from urban areas.

Table 5: Distribution of patients by residence

Residence	No. of Cases (N)	Percentage (%)
Rural	23	57.5
Urban	17	42.5
Total	40	100.0

Graph 5: Distribution of patients by residence



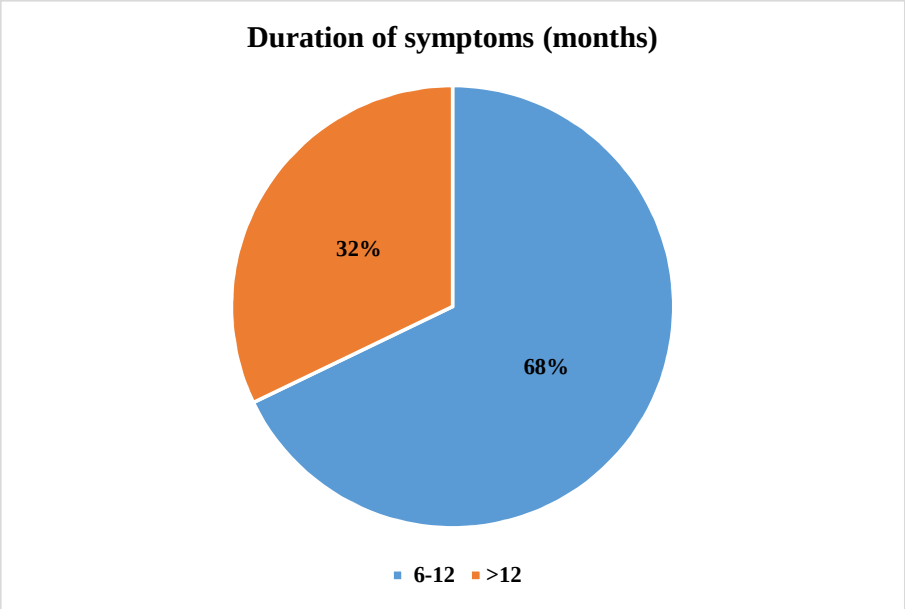
6. Duration of symptoms

In this study, majority of patients, i.e. 52.5% (n=21) had duration of symptoms ranging between 6-12 months while the remaining, i.e. 47.5% (n=19) had duration of symptoms lasting for more than 12 months. Minimum and maximum duration of symptoms observed were 6 months and 24 months respectively.

Table 6: Distribution of patients by duration of symptoms (months)

Duration of symptoms	No. of Cases (N)	Percentage (%)
6-12	19	47.5
>12	21	52.5
Total	40	100.0
Mean ± SD (Range)	14.83±6.41 (6-24 months)	

Graph 6: Distribution of patients by duration of symptoms (months)



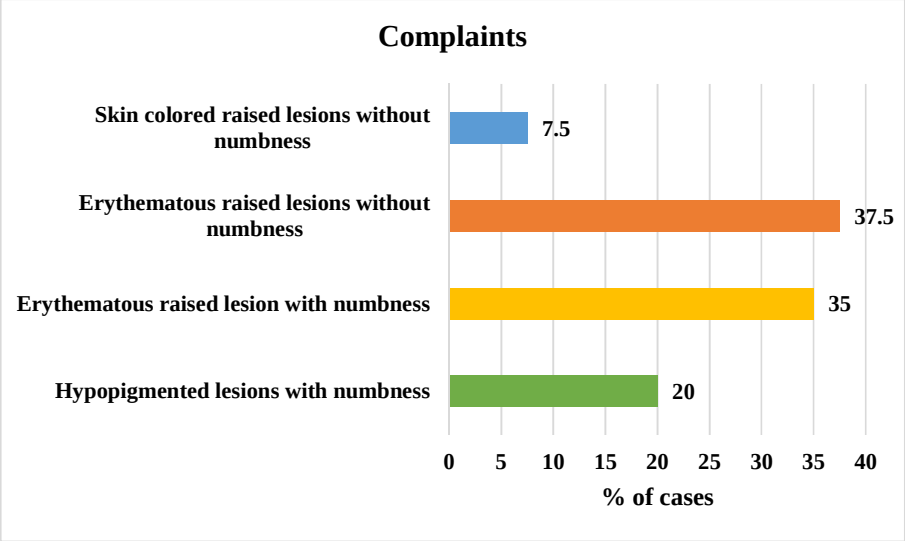
7. Presenting complaints

In this study out of 40 patients, 15 (37.5%) patients had the complaints of erythematous raised lesions without numbness, 14 (35%) patients had erythematous raised lesions with numbness. Numbness and hypopigmented lesions in 8 (20%) patients and skin-colored raised lesions without numbness in 3 (7.5%) patients.

Table 7: Distribution of patients by presenting complaints

Complaints	No. of Cases (N)	Percentage (%)
Hypopigmented lesions with numbness	08	20.0
Erythematous raised lesion with numbness	14	35.0
Erythematous raised lesions without numbness	15	37.5
Skin colored raised lesions without numbness	03	7.5
Total	40	100.0

Graph 7: Distribution of patients by presenting complaints



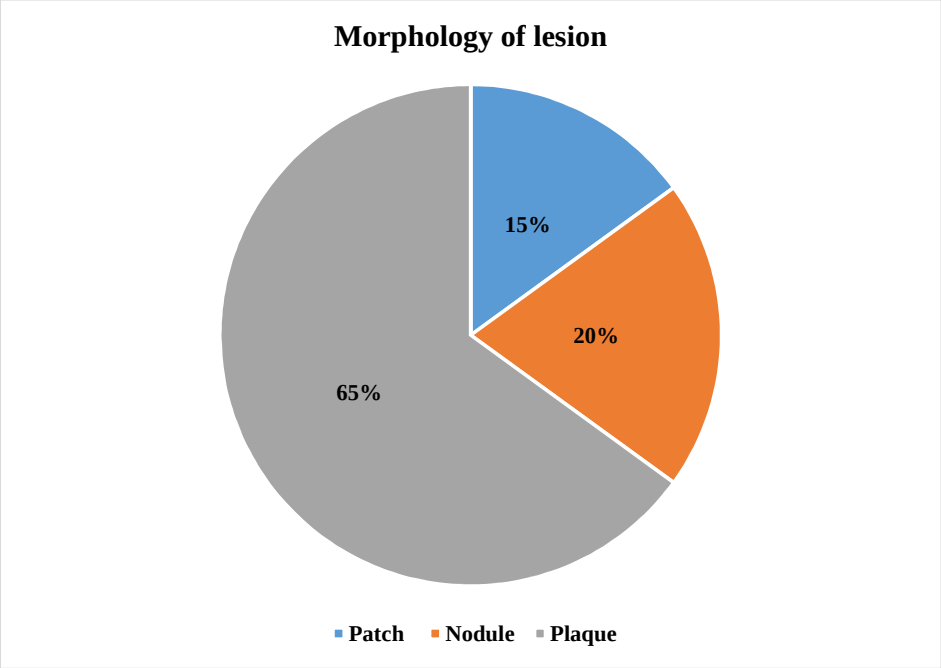
8. Morphology of lesion

In this study majority of patients had plaques on examination (65%). 8% of patients had nodules while 6% had patches.

Table 8: Distribution of patients by morphology of lesion

Morphology of lesion	No. of Cases (N)	Percentage (%)
Patch	06	15.0
Nodule	08	20.0
Plaque	26	65.0
Total	40	100.0

Graph 8: Distribution of patients by morphology of lesion



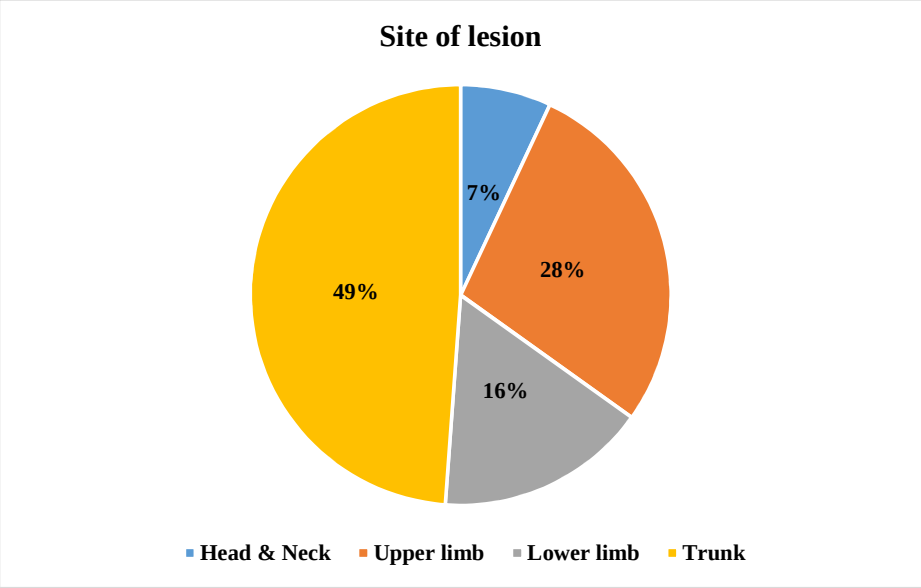
9. Site of lesions

In our study, majority of patients had lesions over trunk, i.e. 21 (52.5 %). 12 patients (30%) had lesions over upper limb, 7 (17.5%) cases had lesions on lower limbs and 3 cases (7.5%) on the head and neck.

Table 9: Distribution of patients by site of lesion

Site of lesion	No. of Cases (N)	Percentage (%)
Head and Neck	03	7.5
Upper limb	12	30.0
Lower limb	07	17.5
Trunk	21	52.5
Total	40	100.0

Graph 9: Distribution of patients by site of lesion



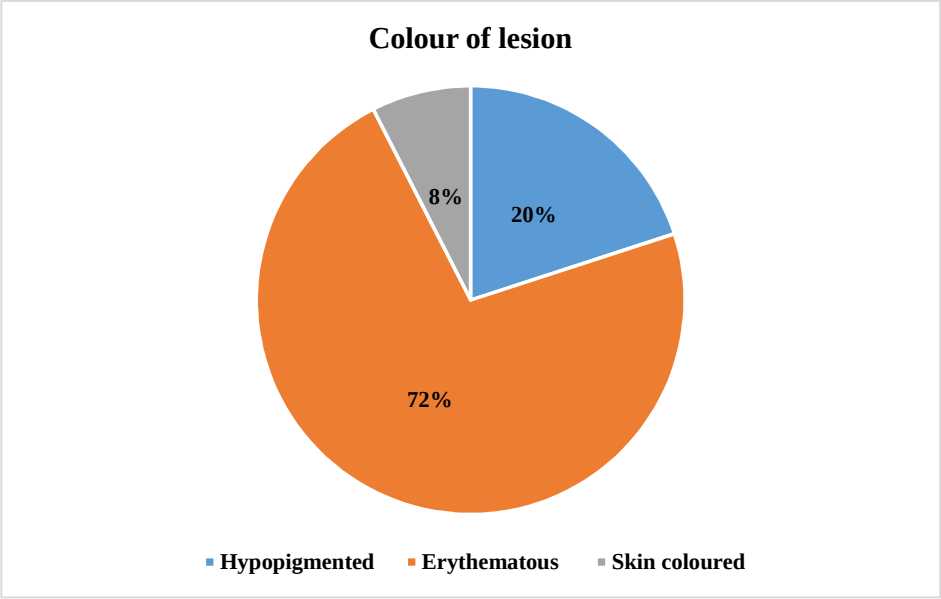
10. Color of lesion

In this study, majority of patients i.e., 72.5% presented with erythematous lesions while 20% and 7.5% patients presented with hypopigmented and skin colored lesions respectively.

Table 10: Distribution of patients by color of lesion

Color of lesion	No. of Cases (N)	Percentage (%)
Hypopigmented	08	20.0
Erythematous	29	72.5
Skin coloured	03	7.5
Total	40	100.0

Graph 10: Distribution of patients by color of lesion



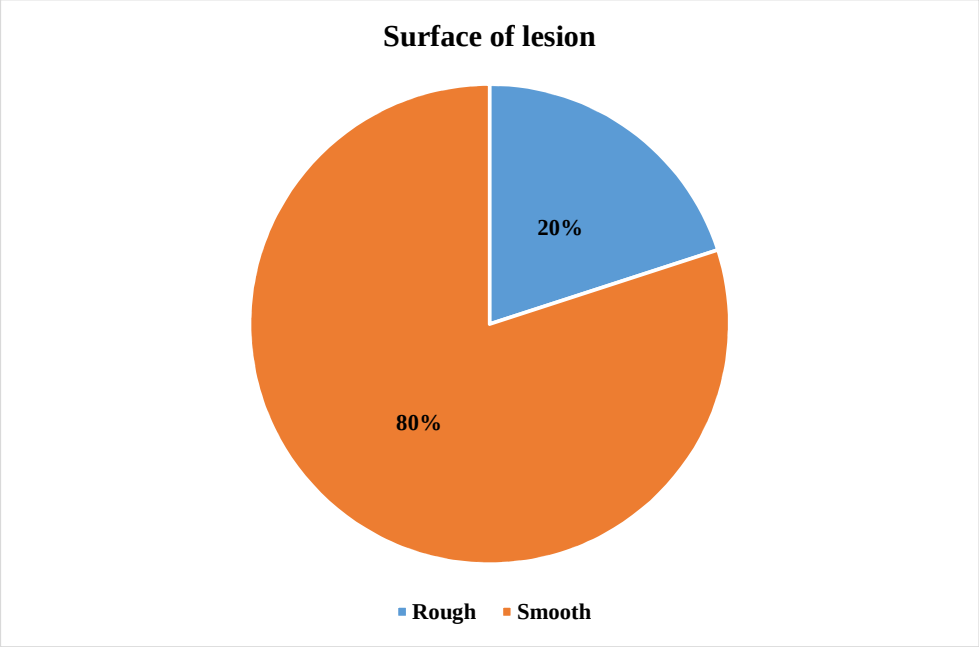
11. Surface of lesion

In this study, 80% of cases had smooth surface over the lesions while the remaining 20% presented with lesions which had rough surface.

Table 11: Distribution of patients by surface of lesion

Surface of lesions	No. of Cases (N)	Percentage (%)
Rough	08	20.0
Smooth	32	80.0
Total	40	100.0

Graph 11: Distribution of patients by surface of lesion



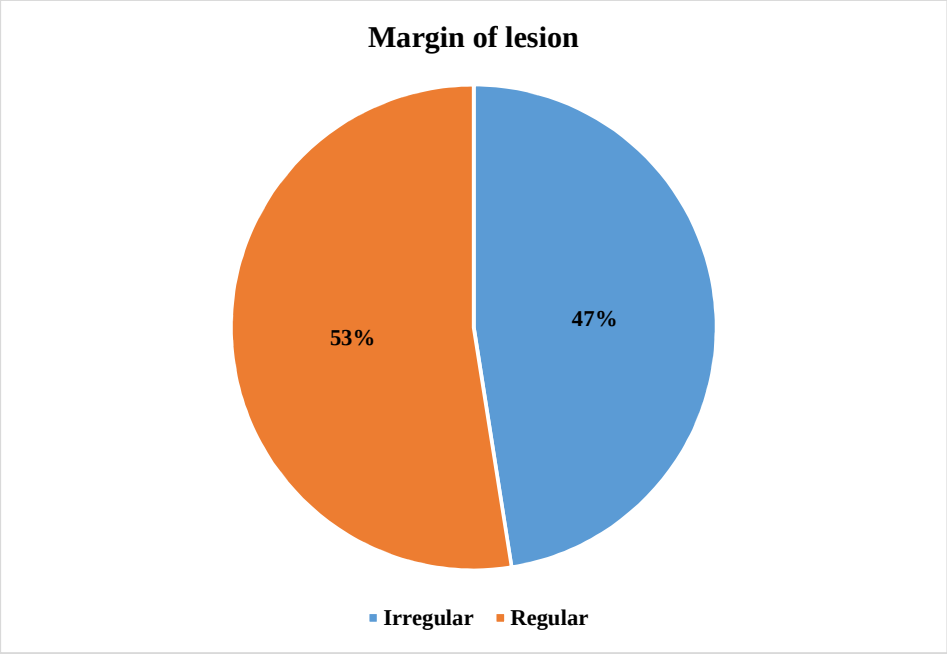
12. Margin of lesion

In this study, 52.5% patients presented with lesions with regular margins while the remaining 47.5% patients had lesions with irregular margins.

Table 12: Distribution of patients by margin of lesion

Margin of lesions	No. of Cases (N)	Percentage (%)
Irregular	19	47.5
Regular	21	52.5
Total	40	100.0

Graph 12: Distribution of patients by margin of lesion



13. Clinical features

In this study, all patients of borderline tuberculoid and tuberculoid leprosy had hypoesthesia over the lesions which corresponded to perineural involvement by granuloma on histopathology seen in these spectrums.

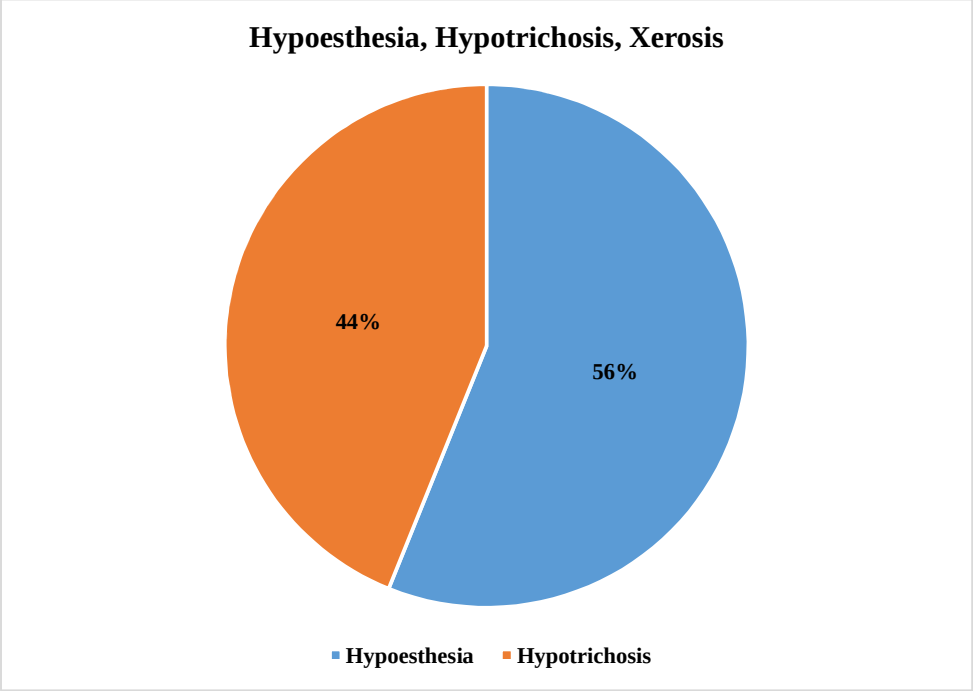
All cases of tuberculoid leprosy and 92.9% of borderline tuberculoid leprosy cases had hypotrichosis over the lesions which corresponded to loss of hair follicles seen on dermoscopy and peri appendageal granuloma on histopathology seen in these spectrums.

76.9% cases of borderline lepromatous leprosy cases had xerosis over the lesions which was seen as characteristic white scaling on dermoscopy.

Table 13: Clinical features seen in different clinical spectrum of leprosy

Feature	Borderline Lepromatous (N=13)	Borderline Tuberculoid (N=14)	Histoid leprosy (N=03)	Lepromatous leprosy (N=05)	Tuberculoid leprosy (N=05)	Total (N=40)
Hypoesthesia	03(23.1)	14(100)	01(33.3)	0	05(100)	23(57.5)
Hypotrichosis	0	13(92.9)	0	0	05(100)	18(45)
Xerosis	10(76.9)	0	0	01(20)	01(20)	12(30)

Graph 13: Clinical features seen in different clinical spectrum of leprosy



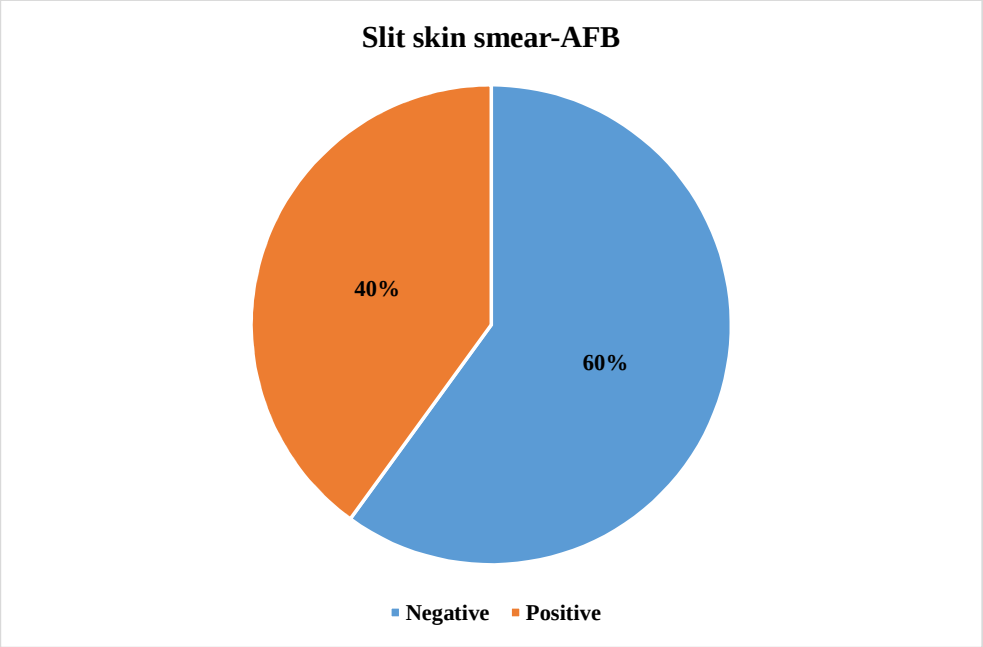
14. Slit skin smear (SSS) for AFB

In this study, 24 cases (60%) showed SSS negative for AFB whereas the remaining 16 cases (40%) showed SSS positive for AFB.

Table 14: Distribution of patients by slit skin smear results for AFB

SSS	No. of Cases (N)	Percentage (%)
Negative	24	60.0
Positive	16	40.0
Total	40	100.0

Graph 14: Distribution of patients by slit skin smear results for AFB



15. Clinical diagnosis

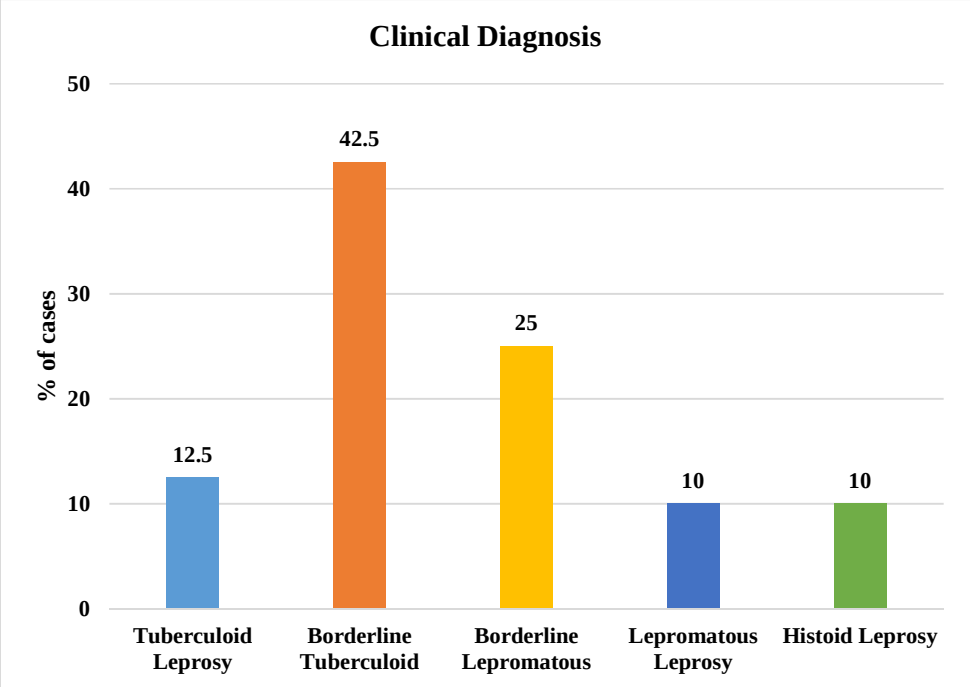
In this study, out of the 40 patients, 17 cases (42.5%) were diagnosed clinically as Borderline Tuberculoid (BT), 10 cases (25%) were clinically diagnosed as Borderline Lepromatous (BL), 5 cases (12.5%) were clinically of Tuberculoid leprosy (TT), 4 cases (10%) were clinically diagnosed as Lepromatous leprosy (LL) and another 4 cases (10%) were diagnosed clinically as histoid leprosy.

Clinical diagnosis was done on the basis of morphology and distribution of lesions and other features such as nerve involvement (hypoesthesia), loss of hair follicles over lesion etc. Histoid leprosy was an addition in this study alongside the spectrum of Ridley Jopling classification. There were no cases of mid borderline leprosy in our study.

Table 15: Distribution of patients by clinical diagnosis

Clinical Diagnosis	No. of Cases (N)	Percentage (%)
Tuberculoid Leprosy	05	12.5
Borderline Tuberculoid	17	42.5
Borderline Lepromatous	10	25.0
Lepromatous Leprosy	04	10.0
Histoid Leprosy	04	10.0
Total	40	100.0

Graph 15: Distribution of patients by clinical diagnosis



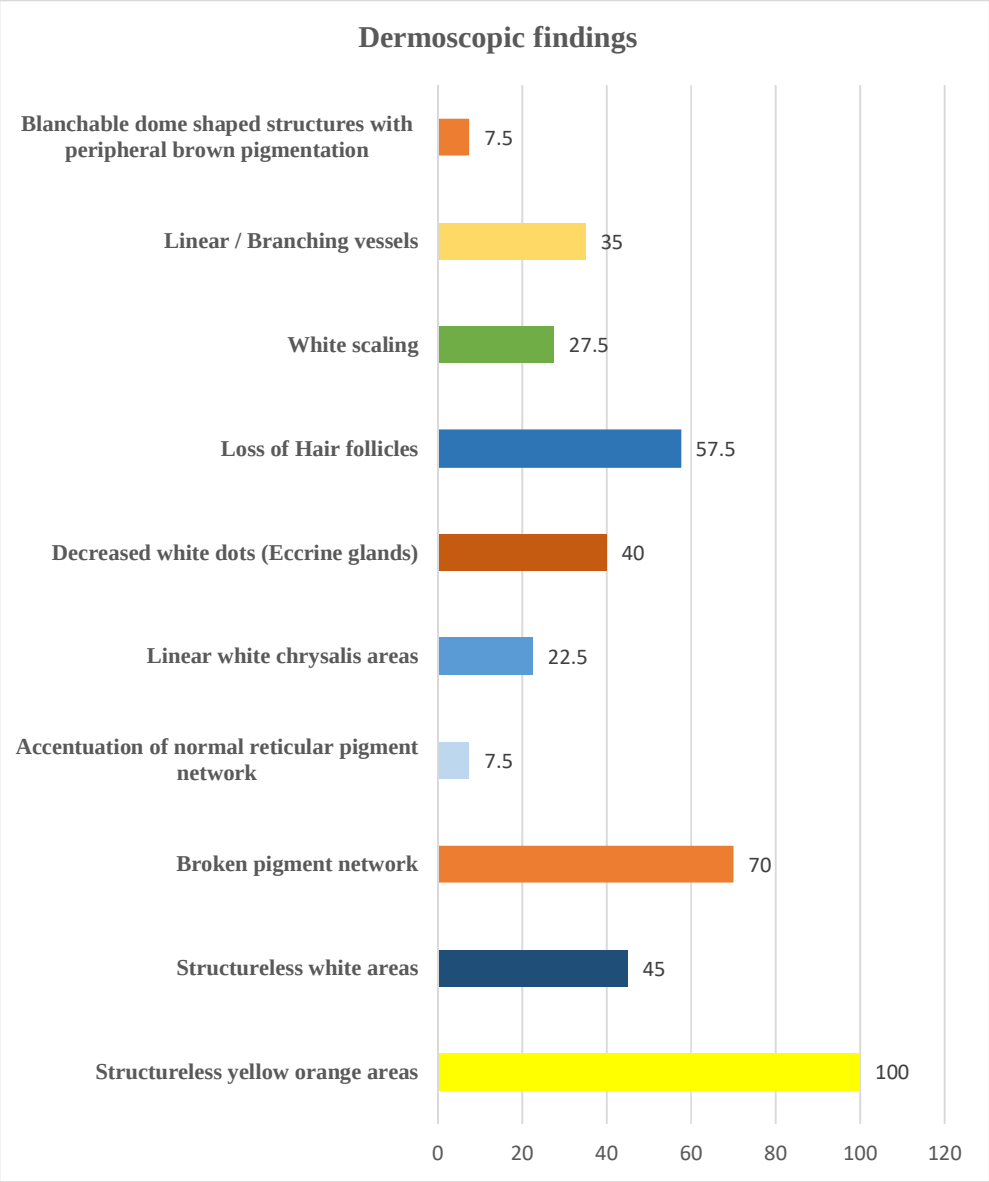
16. Dermatoscopic findings

On dermoscopy, structureless yellow areas were seen consistently in all cases of leprosy. The following table summarizes the different findings on dermoscopic examination.

Table 16: Distribution of cases by dermatoscopic findings

Dermatoscopic findings	No. of Cases (N)	Percentage (%)
Structureless yellow orange areas	40	100.0
Structureless white areas	18	45.0
Broken pigment network	28	70.0
Accentuation of normal reticular pigment network	03	7.5
Linear white chrysalis areas	09	22.5
Decreased white dots (eccrine glands)	16	40.0
Loss of hair follicles	23	57.5
White scaling	11	27.5
Linear / branching vessels	14	35.0
Blanchable dome shaped structures with peripheral brown pigmentation	03	7.5

Graph 16: Distribution of cases by dermatoscopic findings



17. Histopathological findings

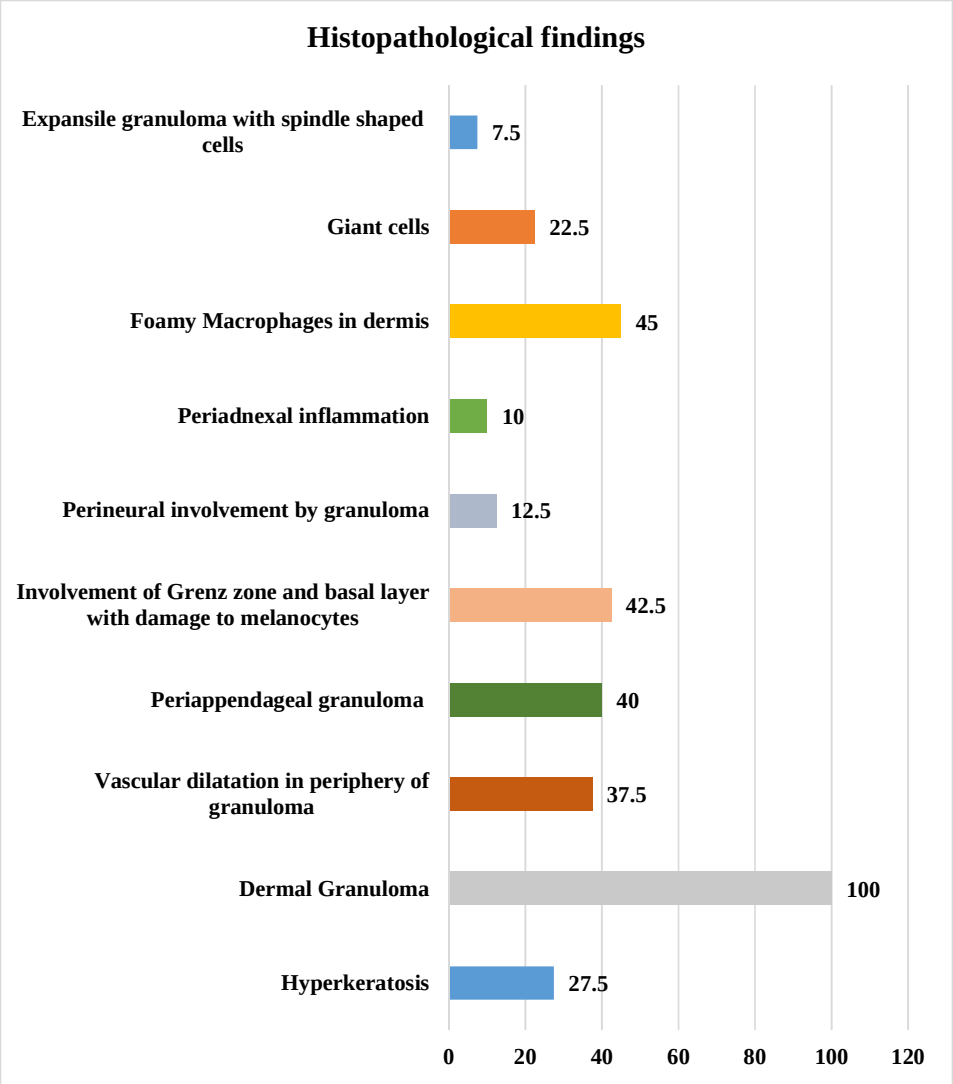
On histopathological examination, dermal granulomas were seen in all the cases (n=40).

The following table summarizes the different findings seen on histopathological examination.

Table 17: Distribution of cases by histopathological findings

Histopathological findings	No. of Cases (N)	Percentage (%)
Hyperkeratosis	11	27.5
Dermal granuloma	40	100.0
Vascular dilatation in periphery of granuloma	15	37.5
Peri appendageal granuloma	16	40.0
Involvement of Grenz zone and basal layer with damage to melanocytes	17	42.5
Perineural involvement by granuloma	05	12.5
Peri adnexal inflammation	04	10.0
Foamy macrophages in dermis	18	45.0
Giant cells	09	22.5
Expansile granuloma with spindle shaped cells	03	7.5

Graph 17: Distribution of cases by histopathological findings



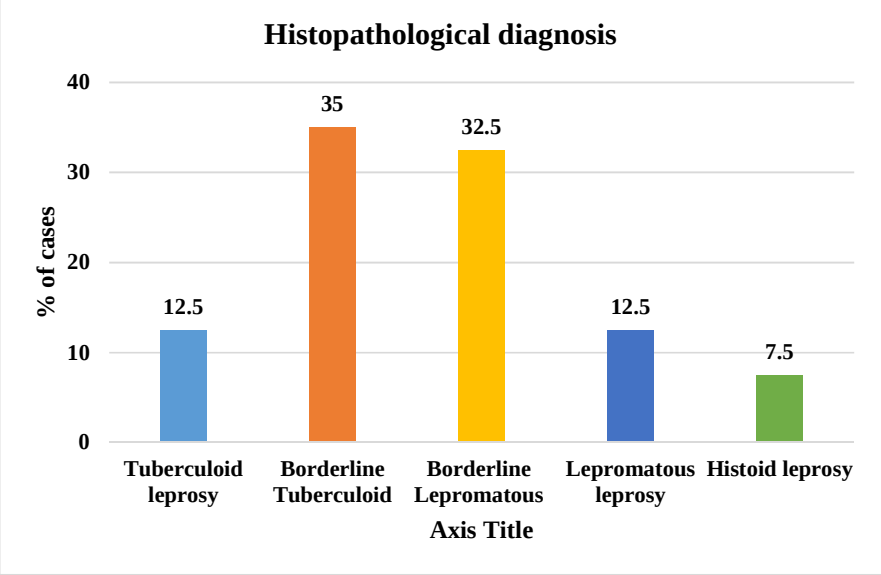
18. Histopathological diagnosis

In our study, 14 cases (35%) were diagnosed as Borderline Tuberculoid (BT) leprosy, 13 cases (32.5%) as Borderline Lepromatous (BL) leprosy, 5 cases (12.5%) as Tuberculoid (TT) leprosy, 5 cases (12.5%) as Lepromatous (LL) leprosy and 3 cases (7.5%) as histoid leprosy. There were no cases of mid borderline leprosy in our study.

Table 18: Distribution of cases by histopathological diagnosis

Histopathological diagnosis	No. of Cases (N)	Percentage (%)
Tuberculoid leprosy	05	12.5
Borderline Tuberculoid	14	35.0
Borderline Lepromatous	13	32.5
Lepromatous leprosy	05	12.5
Histoid leprosy	03	7.5

Graph 18: Distribution of cases by histopathological diagnosis



19. Agreement of histopathological diagnosis with clinical diagnosis

Out of the 17 clinically diagnosed BT leprosy cases, 3 cases were diagnosed as BL on histopathology. Out of the 4 cases of clinically diagnosed histoid leprosy, 1 case was diagnosed as LL on histopathology.

BL, TT and LL showed complete concordance between clinical and histopathological diagnosis.

In this study, highest agreement between clinical and histopathological diagnosis were seen in borderline lepromatous, tuberculoid and lepromatous leprosy of 100% while lowest agreement was seen in histoid leprosy of 75%.

Sometimes, there is disagreement between clinical and histopathological diagnosis as the clinical diagnosis was made on the basis of Ridley Jopling classification before the histopathological diagnosis was made.

Table No.19: Agreement of histopathological diagnosis with clinical diagnosis

Clinical Diagnosis	**Histopathological Diagnosis**						
	BT	BL	TT	LL	Histoid	Total	**% of Agreement**
BT	14	3	0	0	0	17	82.3
BL	0	10	0	0	0	10	100
TT	0	0	5	0	0	5	100
LL	0	0	0	4	0	4	100
Histoid	0	0	0	1	3	4	75
Total	14	13	5	5	3	40	

20. Association of histopathological findings with clinical spectrum

As shown in the table below, significant association ($p<0.05\%$ by Fisher's exact test), was found between presence of various histopathological features with different clinical spectrum of leprosy.

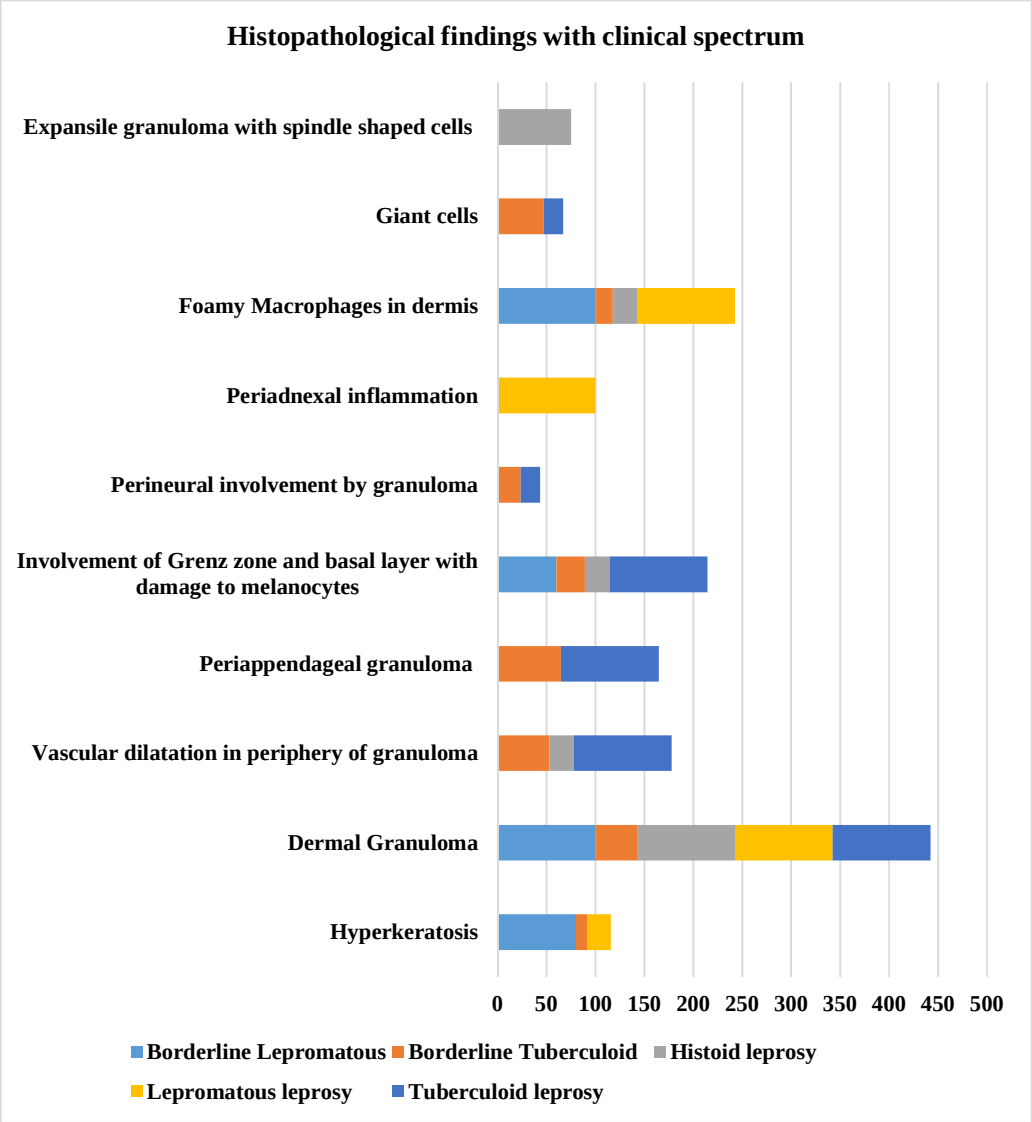
- Dermal granulomas are seen in all cases of leprosy (100%).
- Hyperkeratosis is seen mainly in borderline lepromatous leprosy (80% , n=8)
- Vascular dilatation in periphery of granuloma is seen in all cases of tuberculoid leprosy (100%) and in 52.9% cases of borderline tuberculoid leprosy cases.
- Peri appendageal granuloma is seen in all cases of tuberculoid leprosy (100%) and in 64.7% cases of borderline tuberculoid leprosy.
- Peri adnexal inflammation was seen in all cases of lepromatous leprosy (100%).
- Involvement of Grenz zone and basal layer with damage to melanocytes was seen in all cases of tuberculoid leprosy (100%) and 60% cases of borderline lepromatous leprosy.
- Foamy macrophages were seen in all cases of lepromatous (100%) and borderline lepromatous leprosy (100%).
- Expansile granuloma with spindle shaped cells was seen in 75% (n=3) cases of histoid leprosy.

Table No.20: Association of histopathological findings with clinical spectrum

Histopathological findings	Borderline lepromatous (N=10)	Borderline tuberculoid (N=17)	Histoid leprosy (N=04)	Lepromatous leprosy (N=04)	Tuberculoid leprosy (N=05)	Total	P value
Hyperkeratosis	**08(80)**	02(11.8)	0	01(25)	0	11(27.5)	0.001*
Dermal Granuloma	10(100)	17(42.5)	04(100)	04(100)	05(100)	40(100)	-
Vascular dilatation in periphery of granuloma	0	**09(52.9)**	01(25)	0	**05(100)**	15(37.5)	0.001*
Periappendageal granuloma	0	**11(64.7)**	0	0	**05(100)**	16(40)	0.001*
Involvement of Grenz zone and basal layer with damage to melanocytes	**06(60)**	05(29.4)	01(25)	0	**05(100)**	17(42.5)	0.009*
Perineural involvement by granuloma	0	**04(23.5)**	0	0	**01(20)**	05(12.5)	0.374
Periadnexal inflammation	0	0	0	**04(100)**	0	04(10)	0.001*
Foamy macrophages in dermis	**10(100)**	03(17.6)	01(25)	**04(100)**	0	18(45)	0.001*
Giant cells	0	**08(47.1)**	0	0	01(20)	09(22.5)	0.024
Expansile granuloma with spindle shaped cells	0	0	**03(75)**	0	0	03(7.5)	0.001*

*statistically significant ($p<0.05$, Fisher Exact test)

Graph 19: Association of histopathological findings with clinical spectrum



21. Association of dermoscopic findings and histopathological findings

As shown by the table below, significant association (p<0.05%) is seen between various dermoscopic features and histopathological findings.

Table 21: Association of dermatoscopic findings and histopathological findings

Dermatoscopic findings	Histopathological findings	Agreement	P value
Structureless yellow orange areas (40)	Dermal granuloma (40)	(40 cases) 100%	-
Structureless white areas (18)	Dermal granuloma (40)	(18 cases) 100%	-
Broken pigment network (28)	Involvement of Grenz zone and basal layer with damage to melanocytes (17)	(15 cases) 53.6%	0.032*
Linear / branching vessels (14)	Vascular dilatation in periphery of granuloma (15)	(14 cases) 100%	0.001*
Decreased white dots (eccrine glands) (16)	Peri appendageal granuloma (16)	(14 cases) 87.5%	0.001*
Loss of hair follicles (23)	Peri appendageal granuloma (16)	(16 cases) 100%	0.001*
White scaling (11)	Hyperkeratosis (11)	(11 cases) 100%	0.001*
Blanchable dome shaped structures with peripheral brown pigmentation (03)	Expansile granuloma with spindle shaped cells (03)	(02 cases) 66.7%	0.011*

*statistically significant (p<0.05, Fisher Exact test)

Table 22: Association of dermatoscopic findings with clinical spectrum

Dermatoscopic findings	Borderline Lepromatous (N=13)	Borderline Tuberculoid (N=14)	Histoid leprosy (N=03)	Lepromatous leprosy (N=05)	Tuberculoid leprosy (N=05)	Total (N=40)	P value
Structureless yellow orange areas	13(100)	14(100)	03(100)	05(100)	05(100)	40(100)	-
Structureless white areas	05(38.5)	**10(71.4)**	01(33.3)	01(20)	01(20)	18(45)	0.150
Broken pigment network	09(69.2)	**13(92.9)**	01(33.3)	0	**05(100)**	28(70)	0.001*
Accentuation of normal reticular pigment network	0	0	0	**03(60)**	0	03(7.5)	0.002*
Linear white chrysalis areas	**09(69.2)**	0	0	0	0	09(22.5)	0.001*
Decreased white dots (eccrine glands)	0	**11(78.6)**	0	0	**05(100)**	16(40)	0.001*
Loss of hair follicles	01(7.7)	**13(92.9)**	0	**04(80)**	**05(100)**	23(57.5)	0.001*
White scaling	03(23.07)	3(21.4)	0	**04(80)**	1 (20)	11(27.5)	0.001*
Linear / branching vessels	0	**08(57.1)**	01(33.3)	0	**05(100)**	14(35)	0.001*
Blanchable dome shaped structures with peripheral brown pigmentation	0	01(7.1)	**02(66.7)**	0	0	03(7.5)	0.019*

*statistically significant ($p<0.05$, Fisher Exact test)

22. Association of dermatoscopic findings with clinical spectrum and histopathology

As per tables 21 and 22, significant association ($p<0.05$ by Fisher's Exact test), was found between various dermatoscopic features with different clinical spectrum of leprosy as well as with histopathology.

Structureless yellow orange areas were seen on dermoscopy in all spectrum of leprosy (100%, n=40) which corresponded to the presence of dermal granulomas on histopathology in all the cases (100% agreement).

Structureless white areas were mainly seen in borderline tuberculoid leprosy cases (71.4%, n= 10) and in few cases of other spectrum of leprosy which was not statistically significant. However, there was 100% agreement between presence of structureless white areas on dermoscopy with the presence of dermal granulomas on histopathology.

Broken pigment network was seen in 70% of all cases of leprosy, out of which it was mainly seen in tuberculoid spectrum (100%) and in borderline tuberculoid spectrum (92.9%). There was a statistically significant agreement of 53.6% between the presence of broken pigment network on dermoscopy and involvement of Grenz zone and basal layer with damage to melanocytes on histopathology.

Accentuation of normal reticular pigment network was seen only in lepromatous leprosy (n=3,60%). However corresponding increase in basal layer melanin was not seen on histopathology.

Linear white chrysalis areas were seen mainly in borderline lepromatous leprosy (n=9, 69.2%).

Decreased white dots (eccrine glands) was seen in all cases of tuberculoid leprosy (100%) and in 78.6% cases of borderline tuberculoid leprosy. This corresponded to the presence of peri appendageal granuloma leading to destruction of eccrine glands on histopathology with an agreement of 87.5%.

Loss of hair follicles was seen in all cases of tuberculoid leprosy (100%) and in 92.9% and 80% cases of borderline tuberculoid and lepromatous leprosy respectively. This had an 100% agreement with the presence of peri appendageal granuloma leading to loss of hair follicles on histopathology.

White scaling was seen in 80% cases of lepromatous leprosy. This corresponded to the presence of dry xerotic skin seen clinically in these patients and resulting hyperkeratosis on histopathology with an agreement of 100%.

Linear branching vessels were seen in all cases of tuberculoid spectrum (100%) and in 57% cases of borderline tuberculoid spectrum. This corresponded to vascular dilatation in the periphery of granuloma on histopathology with an agreement of 100%.

Histoid leprosy (n=2, 66.7%) showed blanchable dome shaped structures with peripheral brown pigmentation. This corresponded to the presence of expansile granuloma with spindle shaped cells on histopathology with an agreement of 66.7%.

23. Sensitivity and Specificity of dermoscopic impression versus histopathological diagnosis

Dermoscopy could identify all cases of tuberculoid leprosy resulting in 100% sensitivity and specificity.

For BT leprosy, dermoscopy showed a sensitivity of 100% and specificity of 88.45%.

For BL leprosy, dermoscopy showed a sensitivity of 76.92% and specificity of 100%.

Dermoscopy showed a sensitivity of 80% and a specificity of 100% for lepromatous leprosy.

For histoid leprosy, dermoscopy showed 100% sensitivity and a specificity of 97.3%.

Table 23. Sensitivity and Specificity of dermoscopic impression versus histopathological diagnosis (Gold standard)

	Sensitivity **Value (%) (95%CI)**	**Specificity** **Value (%) (95%CI)**	**PPV** **Value (%) (95%CI)**	**NPV** **Value (%) (95%CI)**	**Accuracy** **Value (%) (95%CI)**
Tuberculoid leprosy	100(47.82-100)	100(90-100)	100	100	100(91.19-100)
Borderline Tuberculoid	100(76.84-100)	88.46(69.85-97.55)	82.35(61.88-93.12)	100	92.50(79.61-98.43)
Borderline Lepromatous	76.92(46.19-94.96)	100(87.23-100)	100	90(76.94-96.04)	92.50(79.61-98.43)
Lepromatous leprosy	80(28.36-99.49)	100(90-100)	100	97.22(85.84-99.51)	97.50(86.84-99.94)
Histoid leprosy	100(29.24-100)	97.30(85.84-99.93)	75(30.26-95.40)	100	97.50(86.84-99.94)

DISCUSSION

Our study was a hospital based cross sectional study conducted in the department of Dermatology, Venereology and Leprosy, KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi over a period of one year from January 2021 to December 2021. The aim of the study was to describe the dermatoscopic findings in different spectrum of leprosy and to associate these findings with the clinical spectrum and histopathological findings of leprosy.

A total of 40 patients of clinically diagnosed treatment naïve leprosy fulfilling all inclusion and exclusion criteria were included in the study.

After obtaining consent, all patients underwent a detailed clinical examination followed by dermoscopic examination of the most characteristic lesion (DermLite DL4,10x contact polarized mode). The same lesion was biopsied and sent for histopathological analysis by H & E staining. Records were maintained and analysed.

Very few studies have been carried out associating dermoscopic features of leprosy with the clinical spectrum and histopathological findings. Our study when compared to other similar studies had many common findings and a few findings in contrast.

In our study we recruited 40 treatment naïve cases of leprosy as upon treatment, the dermoscopic features may show changes such as missing yellowish orange hue and vascular structures. This was similar to a study conducted by Vinay K et al.[69] where 30 treatment naïve cases of leprosy were recruited.

This was in contrast to a study conducted by Mohta A et al.[70] and Chopra et al.[71] wherein along with new cases of leprosy, patients taking treatment for a period of less than 6 months were also included. In a study conducted by Bhatia et al.[74] the treatment status of the patients was not taken into consideration.

In our study there were 40 subjects of treatment naïve leprosy with mean age of 43.43±10.09 years. This was near similar to the study performed by Vinay K et al.[69], wherein 30 subjects of treatment naïve leprosy were recruited with a mean age of 42 years.

There was a male preponderance in our study, i.e. out of 40 patients, there were 27 males (67.5%) and 13 females (32.5%) with male to female ratio of 2.07:1. This was found to be similar to the study conducted by Vinay K et al.[69] which included 21 males and 9 females, i.e. a male predominance of 70%. Similar male predominance of 64% was found in a study conducted by Chopra et al.[71]

In our study we also evaluated the occupation, socioeconomic status and residence of the patients which was not done in other studies. We found that majority of patients were farmers (40%) and were from low-income group (57.5%) residing in rural areas (57%) which shows that leprosy is prevalent in such populations.

In our study, duration of symptoms ranged between 6-24 months with a mean duration of 14.83±6.41months. This was near similar to the study conducted by Bhatia et al.[74] where mean duration of symptoms was 12 months.

In our study, site, morphology, color of lesion, surface and margin of lesion were also evaluated which was not done in other studies. Most common site of the characteristic lesion was the trunk (52.5%), plaque being the most common

morphology (65%) which were mostly erythematous (72.5%) with smooth surface (80%) and regular margins (52.5%).

After clinical and histopathological examination, the subjects were classified into different spectrum of leprosy according to Ridley Jopling classification. Our study showed that majority of the subjects (35%, n=14) had borderline tuberculoid leprosy followed by borderline lepromatous leprosy (32.5%, n=13), tuberculoid leprosy (12.5%, n=5), lepromatous leprosy (12.5%, n=5) and histoid leprosy (7.5%, n=3). Histoid leprosy was an additional spectrum that was included in our study. No mid borderline cases were found during our period of study.

In this study, highest agreement between clinical and histopathological diagnosis were seen in borderline lepromatous, tuberculoid and lepromatous leprosy of 100% while lowest agreement was seen in histoid leprosy of 75%.

Sometimes there is disagreement between clinical and histopathological diagnosis as the clinical diagnosis was made on the basis of Ridley Jopling classification before the histopathological diagnosis was made.

This was similar to studies conducted by Vinay et al.[69] , Mohta A et al.[70], Chopra et al.[71], Kakitha et al.[73] and Bhatia et al.[74], where borderline tuberculoid was the most common spectrum seen.

In the study by Mohta A et al.[70] borderline tuberculoid consisted of 39% cases, followed by borderline lepromatous (26%), followed by lepromatous leprosy (23%), tuberculoid leprosy (5.4%) and histoid leprosy (5.4%). However, lepra reactions were also taken into consideration in contrast to our study.

In the study conducted by Chopra et al.[71], 50 patients were recruited, 10% of which were of borderline tuberculoid leprosy and another 10% of lepromatous leprosy. However, 10% patients each of type 1 and type 2 lepra reactions and another 10% patients of clofazimine induced hyperpigmentation were also included in contrast to our study.

In the study conducted by Vinay K et al.[69], 40% patients were of borderline tuberculoid, 26% were of borderline lepromatous leprosy, 20% were of lepromatous leprosy and 6% each of tuberculoid and histoid leprosy.

In the study conducted by Kakitha et al.[73], 45.4% cases belonged to borderline tuberculoid leprosy, 18.2% each of borderline lepromatous and lepromatous leprosy, 12.1% belonged to tuberculoid leprosy. No patients of histoid leprosy were recruited in contrast to our study.

In the study conducted by Bhatia et al.[74], 36% cases were of borderline tuberculoid spectrum, 26% were of borderline lepromatous, 20% cases were of lepromatous leprosy, 10% of tuberculoid spectrum and 6% of histoid leprosy. However, lepra reactions were also included in the study in contrast to our study.

Dermoscopy revealed structureless yellow orange areas (n=40,100%) which corresponded to the presence of dermal granuloma on histopathology (100% agreement), across all spectrum of leprosy.

This was in agreement with the studies conducted by Vinay K et a.[69], Mohta A et al.[70], Chopra et al.[71] and Bhatia et al.[74] where similar yellowish orange structureless areas were seen on dermoscopy corresponding to dermal granuloma on

histopathology. However, in these studies, borderline lepromatous spectrum did not show these structureless areas in contrast to our study.

Also, this finding in our study is in contrast with the study conducted by Kakitha et al.[73] where structureless areas were seen which were not yellow orange.

Dermoscopy of borderline tuberculoid leprosy revealed structureless white areas (71.4%) corresponding to dermal granulomas seen on histopathology (100% agreement) , linear/branching vessels (57.1%) corresponding to vascular dilatation in the periphery of granuloma on histopathology (100% agreement), broken pigment network (92.9%) corresponding to involvement of Grenz zone and basal layer with damage to melanocytes on histopathology (53.6% agreement), decreased white dots (eccrine glands) (78.6%) and loss of hair follicles(92.9%) corresponding to peri appendageal involvement by granuloma on histopathology (87.5% agreement). The dermoscopic findings are significantly associated with the corresponding histopathological features ($p = 0.001$). These findings are in agreement with study conducted by Mohta A et al.[70], Chopra et al.[71] and Bhatia et al.[74] where in addition to these features yellow dots and globules were also seen corresponding to dilatation of pilosebaceous units on histopathology. This feature was not observed in our study. The findings in our study were also in agreement with study conducted by Vinay K et al.[69] where structureless yellowish-orange areas were seen along with branching and anastomosing vessels, paucity of appendageal structures and diminished pigment network.

Dermoscopy of borderline lepromatous leprosy lesions revealed broken pigment network (69.2%) corresponding to involvement of basal layer by the granuloma with damage to melanocytes on histopathology. Linear white chrysalis like areas (69.2%)

were present which was seen specifically in this spectrum. Also, a relative sparing of appendages was seen.

These findings were in agreement with the study conducted by Mohta A et al.[70] where similar linear white streaks with relative sparing of appendages were present. However, this corresponded to perifollicular hyperkeratosis on histopathology which was not seen in our study. The relative sparing of appendages was in contrast to the study conducted by Vinay K et al.[69] and Bhatia et al.[74] where the same features were present with a contrasting paucity of appendages and hair follicles.

Dermoscopy of all lesions (100%) of tuberculoid leprosy revealed broken pigment network corresponding to involvement of basal layer by the granuloma with damage to melanocytes on histopathology, loss of hair follicles and reduced white dots (eccrine glands) corresponding to peri-appendageal granuloma on histopathology. Linear branching vessels were seen around the structureless yellow orange areas corresponding to vascular dilatation in the periphery of granuloma on histopathology. Hence the dermoscopic features were very similar to the ones seen in borderline tuberculoid spectrum.

These findings were consistent with the studies conducted by Mohta A et al.[70] and Bhatia et al.[74] No tuberculoid leprosy cases were included in the study conducted by Vinay K et al.[69]

In our study, dermoscopy of lepromatous leprosy revealed accentuation of normal reticular pigment network (60%) in contrast to other spectrum of leprosy where broken pigment network is found. Loss of hair follicles was seen in 80% cases.

These findings are consistent with the study conducted by Mohta A et al.[70] and Vinay et al.[69]

In addition, characteristic white scaling was also seen in 80% of cases corresponding to xerotic skin clinically and hyperkeratosis on histopathology which is consistent with the study conducted by Chopra et al.[71] and Bhatia et al.[74]

In our study, dermoscopy of histoid leprosy (66.7%) revealed blanchable dome shaped structures with peripheral brown pigmentation which corresponded to expansile granulomas with spindle shaped cells on histopathology (66.7% agreement).

This was in near similar to the study conducted by Vinay K et al.[69] and Bhatia et al.[74] where dome-shaped yellowish-brown areas with few crown vessels over the margins were seen on dermoscopy of histoid leprosy lesions. However, no crown vessels could be visualized in our study. This could be probably because of the contact mode used in our study which could have caused blanching of the vessels.

It was also consistent with the study conducted by Mohta A et al.[70] where similar dome shaped blanchable structures which were centrally hypopigmented were seen with crown vessels and perilesional hyperpigmentation corresponding to expansile granuloma with spindle shaped cells. Also, central white dots and keratotic plugs in central umbilication with pseudo koebnerization were seen corresponding to transepidermal elimination on histopathology and crystalline lines and shiny white areas corresponding to dermal fibrosis on histopathology. These additional findings were not seen in our study.

There was significant association between features seen on dermoscopy with the clinical spectrum and histopathology ($p < 0.001$) (calculated by Fisher's exact test).

Similar association were present in the studies conducted by Vinay K et al.[69], Mohta A et al.[70], Chopra et al.[71] and Bhatia et al.[74] However in none of the studies, p value was calculated denoting the statistical significance of the association between dermoscopy and clinical spectrum and histopathology. Also, this was in contrast to the study by Gervasio et al.[75] where no significant association was found between dermoscopic features and Ridley Jopling classification. Also, no association of dermoscopy features with histopathological features were found apart from a significant association of structureless areas on dermoscopy with the presence of granulomas on histopathology (100%).

In our study, it was found that dermoscopy could identify all cases of tuberculoid leprosy resulting in 100% sensitivity and specificity. For BT leprosy, dermoscopy showed a sensitivity of 100% and specificity of 88.45%. For BL leprosy, dermoscopy showed a sensitivity of 76.92% and specificity of 100%. Dermoscopy showed a sensitivity of 80% and a specificity of 100% for lepromatous leprosy. For histoid leprosy, dermoscopy showed 100% sensitivity and a specificity of 97.3%.

Our study showed an unarguably high sensitivity and high specificity of dermoscopy hence proving it to be a handy modality which can aid in the diagnosis of leprosy.

CONCLUSION

The study was done to describe the dermatoscopic features in different spectrum of leprosy and also to associate these findings with clinical spectrum and histopathology.

Dermoscopic features of all forms of leprosy associated well with histopathological findings. It was also found to have good diagnostic accuracy.

Not only does dermoscopy aid in diagnosing leprosy but also it helps in diagnosing different spectrum of leprosy. Also, it helps in differentiating it from other granulomatous conditions and pigmentary disorders which present with similar hypopigmented patches.

Dermoscopy can be used as a handy tool to complement histopathological diagnosis which still remains the gold standard in diagnosing leprosy. It can also be used for diagnosis of leprosy in resource poor settings where facilities for histopathology are unavailable and in cases where biopsy could not be done as in facial lesions, children and non-consenting patients.

However, the small sample size (n=40) was the limitation in our study. But in view of the low incidence of leprosy in India leading to dearth in treatment naïve cases, the small sample size of the study can be considered reasonable.

SUMMARY

The present cross-sectional study was conducted among the patients with leprosy attending Dermatology, Venereology and Leprosy OPD at KLE'S Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi during the period of January 2021 to December 2021. All participants underwent dermoscopic examination using DermLite DL4 dermoscope of the most characteristic lesion with findings being noted along with recording of both clinical and dermoscopic images. Skin biopsy was taken from the same site where dermoscopy was done taking all aseptic precautions and sent for analysis. All the data were collected in proforma and entered in excel sheet.

The present study aimed to assess the dermatoscopic features of different spectrum of leprosy and also to associate the findings with clinical spectrum and histopathological findings among the patients.

- In present study total of 40 patients fulfilling inclusion criteria are included with mean age of 43.43±10.09yrs of age.
- Among them, majority of the patients age were ranged from 41-60yrs in 47.5% and 21-40yrs in 45% of the patients.
- Among them, 67.5% were male patients and 32.5% were female patients.
- By occupation, majority were farmers (40%) followed with 25% housewife, 12.5% were shopkeepers and 10% were labourers.
- On assessment of the socioeconomic status of the patients, 57.5% belonged to low income group and 42.5% belonged to moderate income group.

- In the study 57.5% patients were from rural areas and 42.5% were from urban areas.
- The symptoms were present for 6-12 months in 47.5% cases and in 22.5% for more than 12 months of duration.
- On assessment of complaints, the study documented that 37.5% cases had erythematous raised lesion without numbness, 35% had erythematous raised lesion with numbness, 20% had hypo pigmented lesion with numbness and 7.5% had skin colored raised lesion without numbness.
- Morphologically, the lesions presented as plaque in 65%, 20% as nodule and 15% as patch.
- On assessment of site of lesion, 52.5% were on trunk, 30% with upper limb, 17.5% on lower limb and 7.5% with head and neck.
- On assessment of the color of lesion, 72.5% were erythematous, 20% were hypopigmented and 7.5% were skin colored.
- The surface of lesion was found to be smooth in 80% cases and rough in 20% cases.
- The margin of lesion was seen as irregular in 47.5% and regular on 52.5% of the patients.
- In the study, 57.5% had hypoesthesia, 45% had hypotrichosis and 30% had xerosis.
- The split skin smear for AFB showed positive result in 40% and 60% were negative.
- On assessing the clinical diagnosis, 42.5% were borderline tuberculoid, 25% were borderline lepromatous, 12.5% were tuberculoid leprosy, 10% each had lepromatous leprosy and histoid leprosy.

- The dermatoscopic findings were found to be 70% with broken pigment network, 57.5% with loss of hair follicles, 45% with structureless white area, 40% with decreased white dots, 35% with linear or branching vessels, 27.5% with white scaling and 22.5% with linear white chrysalis areas.
- The histopathological findings showed the presence of the foamy macrophages in dermis in 45%, 42.5% with involvement of Grenz zone and basal layer with damage to melanocytes, 40% with peri appendageal granuloma, 37.5% with vascular dilatation in periphery of granuloma, 27.5% showed hyperkeratosis. All the cases showed dermal granuloma.
- Histopathological diagnosis showed 35% with borderline tuberculoid, 32.5% with borderline lepromatous, 12.5% with tuberculoid leprosy, another 12.5% with lepromatous leprosy and 7.5% with histoid leprosy.
- Highest agreement between clinical and histopathological diagnosis were seen in borderline lepromatous, tuberculoid and lepromatous leprosy of 100% while lowest agreement was seen in histoid leprosy of 75%.
- Structureless yellow orange areas were seen on dermoscopy in all spectrum of leprosy (100%, n=40) which corresponded to the presence of dermal granulomas on histopathology in all the cases (100% agreement).
- Structureless white areas were mainly seen in borderline tuberculoid leprosy cases (71.4%, n= 10) and in few cases of other spectrum of leprosy which was not statistically significant. However, there was 100% agreement between presence of structureless white areas on dermoscopy with the presence of dermal granulomas on histopathology.
- Broken pigment network was seen in 70% of all cases of leprosy, out of which it was mainly seen in tuberculoid spectrum (100%) and in borderline

tuberculoid spectrum (92.9%). There was a statistically significant agreement of 53.6% between the presence of broken pigment network on dermoscopy and involvement of Grenz zone and basal layer with damage to melanocytes on histopathology.

- Accentuation of normal reticular pigment network was seen only in Lepromatous leprosy (n=3,60%). However corresponding increase in basal layer melanin was not seen on histopathology.
- Linear white chrysalis areas were seen mainly in borderline lepromatous leprosy (n=9, 69.2%).
- Decreased white dots (eccrine glands) was seen in all cases of tuberculoid leprosy (100%) and in 78.6% cases of borderline tuberculoid leprosy. This corresponded to the presence of peri appendageal granuloma leading to destruction of eccrine glands on histopathology with an agreement of 87.5%.
- Loss of hair follicles was seen in all cases of tuberculoid leprosy (100%) and in 92.9% and 80% cases of borderline tuberculoid and lepromatous leprosy respectively. This had an 100% agreement with the presence of peri appendageal granuloma leading to loss of hair follicles on histopathology.
- White scaling was seen in 80% cases of lepromatous leprosy. This corresponded to the presence of dry xerotic skin seen clinically in these patients and resulting hyperkeratosis on histopathology with an agreement of 100%.
- Linear branching vessels were seen in all cases of tuberculoid spectrum (100%) and in 57% cases of borderline tuberculoid spectrum. This corresponded to vascular dilatation in the periphery of granuloma on histopathology with an agreement of 100%.

- Histoid leprosy (n=2, 66.7%) showed blanchable dome shaped structures with peripheral brown pigmentation. This corresponded to the presence of expansile granuloma with spindle shaped cells on histopathology with an agreement of 66.7%.

- Significant association was found between dermoscopy and clinical spectrum and histopathological findings.

- The diagnostic accuracy of dermoscopy was found to be significant. Dermoscopy could identify all cases of tuberculoid leprosy resulting in 100% sensitivity and specificity. For BT leprosy, dermoscopy showed a sensitivity of 100% and specificity of 88.45%. For BL leprosy, dermoscopy showed a sensitivity of 76.92% and specificity of 100%. Dermoscopy showed a sensitivity of 80% and a specificity of 100% for lepromatous leprosy. For histoid leprosy, dermoscopy showed 100% sensitivity and a specificity of 97.3%.

Hence it can be concluded that dermoscopy acts as an effective tool and aids in diagnosing the different clinical spectrum of leprosy.

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ANNEXURE I - INFORMED CONSENT FORM

I.D.NO. ☐☐☐

Title of the study.: **"A ONE YEAR HOSPITAL BASED CROSS SECTIONAL STUDY OF DERMATOSCOPY IN LEPROSY AND ITS ASSOCIATION WITH CLINICAL SPECTRUM AND HISTOPATHOLOGY AT A TERTIARY CARE CENTRE. "**

The study is conducted _____, Post Graduate (M.D.) student in Dermatology under the guidance of **_____,** Associate Professor, Department of Dermatology, Venereology and Leprosy, JNMC, BELAGAVI.

Respected Sir/Madam,

We invite you to participate in our study as you are eligible for the same. During the study you will be asked some questions in detail regarding your present complaints.

Purpose of the study:

Leprosy lesions can be seen using an instrument called dermatoscope. Hence this study intends to observe those changes/findings using the dermatoscope. Following which confirmation of the diagnosis will be done using biopsy. You are being requested to participate in this study because you have been diagnosed to have leprosy.

Procedure:

Should you choose to participate, you will be asked to give a detailed history of your disease, undergo a physical examination. Following which over your leprosy lesions dermoscopic examination will be done and over a specified lesion skin biopsy will be performed which requires local anaesthesia and 4mm of skin will be taken for the purpose of study after which it will be studied under a light microscope for the confirmation of diagnosis.

Risks and Benefits:

While doing skin biopsy, you may experience slight pain due to the procedure. However, all necessary steps and precautions will be taken to ensure your safety. The result of you taking part in this research would help health care providers towards a better understanding and early diagnosis of this disease, and thus we will be able to provide improved patient care.

Alternatives:

If you decide not to participate in this study, you will still be receiving the usual standard care for your disease.

Privacy and confidentiality:

Your privacy will be respected and all information collected about you during the course of this study will be kept confidential. Your identity will remain undisclosed.

Relations with the Institutional policy:

The J N Medical College will provide, within the limitations of the laws of the State of Karnataka, facilities and medical attention to patients who suffer injuries as a result of participating in this project.

Financial incentives:

You shall not be receiving any payment or any financial incentives for participating in this study.

Authorization to publish results:

The results of this study may be published for scientific purpose or presented to a scientific group. Your identity, however, will be maintained confidential at all times.

Voluntary participation:

Your participation in this study is voluntary. Your decision whether or not to participate will neither affect the care of your current disease, nor your future relations with the doctor or the hospital. In the event if you suffer any physical injury as the result of your participation in this study, you may contact

In case you need further information regarding your rights as a study participant, you may please contact **Dr. HARSHA HEGDE**, chairman of the ethical committee, J N Medical College, Belagavi.

STATEMENT OF CONSENT

I.D.NO: | | | |

I, Mr. /Ms./Mrs. ---------- volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in the study. All the information regarding this study is provided to me and I have understood the same. I have been given the opportunity to ask questions and obtain appropriate answers.

Participant's name:

Signature or left thumb print of participant:

Witness name:

Signature of witness:

Signature of the investigator

ANNEXURE II - PROFORMA

Title : " A one year hospital based cross sectional study of dermatoscopy in leprosy and its association with clinical spectrum and histopathology" at KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi

Case No.

Date

Name

Age

Sex

Occupation

Income

Education level

Address

Consent : I have been explained the nature of my condition in my own language and I understand the possible risks and complications that I may experience during the course of my treatment. I hereby give full consent towards undergoing any diagnostic or therapeutic procedure during the course of my illness, and I understand the possible risks involved. I have no objection towards use of my medical information or photographs for academic and research purposes, knowing that my confidentiality will be maintained at all times.

Signature of left-thumb impression of patient

Date:

I. Presenting complaints (with duration)

a. Change in skin color : Yes/No

b. Loss of sensation: Yes/ No

c. Tingling and numbness : Yes/No

d. Nasal stuffiness : Yes/No

e. Swelling of feet : Yes/No

f. Deformities: Yes/No

g. Other: Yes/No

II. History of present illness

a. Change in skin color : Yes/No

b. Loss of sensation: Yes/No

c. Tingling and numbness : Yes/No

d. Nose changes: Yes/No

e. Swelling of feet : Yes/No

f. Deformities: Yes/No

g. Eye changes : Yes/No

h. Loss of hair and eyebrows: Yes/No

i. Hoarseness : Yes/No

j. Loss of teeth: Yes/No

k. Bone/Joint pain : Yes/No

l. Burning micturition : Yes/No

m. Scrotal swelling: Yes/No

n. Impotence : Yes/No

o. Generalized weakness: Yes/No

p. Constitutional symptoms: Yes/No

q. Any other complaints :

III. Past history

a. Diabetes mellitus : Yes/No

b. Hypertension: Yes/No

c. Asthma : Yes/No

d. Epilepsy: Yes/No

e. HIV : Yes/No

f. Others :

IV. Family history

a. Married / Single

b. Similar complaints in family members: Yes/No

V. Treatment history

a. Has taken anti-leprosy treatment: Yes/No

b. Any other treatment taken : Yes/No

VI. Personal history

a. Diet : Mixed/Vegetarian

b. Appetite: Normal/Decreased/Increased

c. Sleep : Adequate/ Inadequate

d. Bowel and Bladder habits : Regular/Irregular

e. Other habits : Smoking/Alcohol/Tobacco/None

VII. General physical examination

a. Build : Poor/ Moderate/ Well

b. Nourishment: Poor/Moderate/Well

c. Pallor/Icterus/Cyanosis/Clubbing/Lymphadenopathy/Edema

d. Pulse

e. BP

f. Weight

g. Temperature

VIII. Mucocutaneous examination

a. Sites of lesions : Head/Trunk/UL/LL

b. Number of lesions: 1 / 2-5 / >5

c. Type of lesions : Macule/ Patch /Plaque/Nodule/Other

d. Size of lesions : Small/ Medium/ Large/ Variable

e. Surface of lesions : Smooth/ Rough

f. Color of lesions : Hypopigmented / Hyperpigmented / Erythematous

g. Margins: Ill defined / Well defined

h. Distribution: Symmetrical / Asymmetrical

i. Sensation over lesions : Present / Impaired / Absent

j. Hair over lesions : Present / Absent

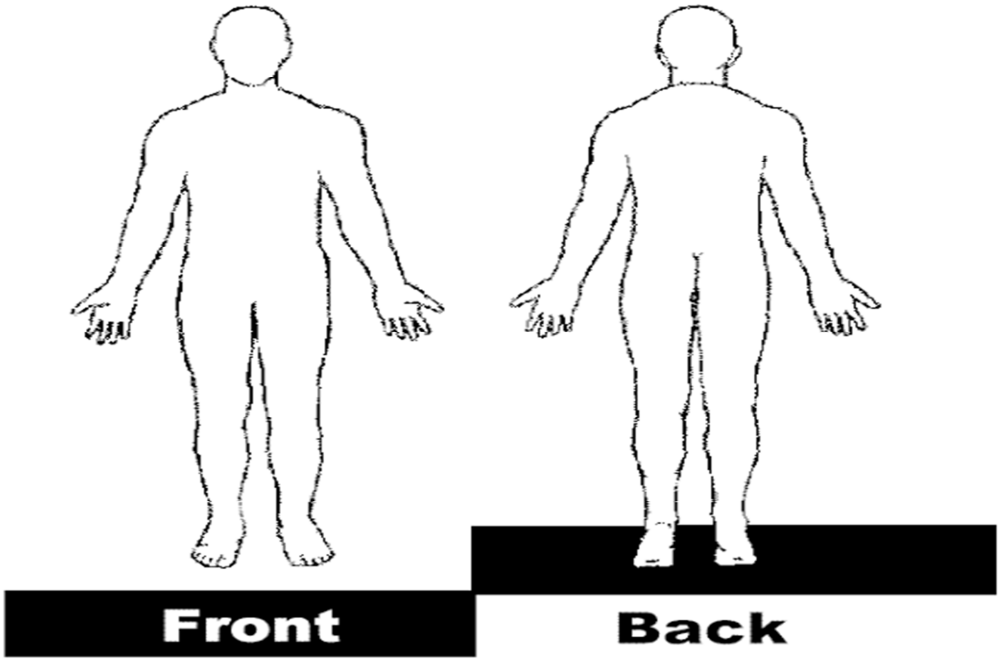
k. Changes over face : Eyebrow loss / Eyelashes lost / Ear thickening/ Nose changes

l. Generalized lymphadenopathy : Present / Absent

m. Swelling of testis : Yes / No

n. Gynaecomastia : Yes / No

o. Other changes: Yes / No



p. Peripheral nerve examination (Normal / Thickened / Tender / Irregular thickening)

Nerve	Right side	Left side
1. Supra-orbital nerve 2. Infra-orbital nerve 3. Greater auricular nerve 4. Radial nerve 5. Ulnar nerve 6. Median nerve 7. Radial cutaneous nerve 8. Common peroneal nerve 9. Posterior tibial nerve 10. Sural nerve 11. Superficial peroneal nerve		

q. Deformities: Claw hand /Claw toe/ Contractures/ Ulcers/ Wrist drop/ Foot drop

h. Eye changes: Conjunctivitis / Iritis/ Lagophthalmos

s. Hair involvement: Yes / No

t. Nail involvement: Yes / No

u. Mucosal involvement: Yes / No

v. If mucosa involved then : Genital / Oral / Anal / Conjunctival

IX. Systemic examination

a. CVS :

b. RS :

c. PA:

d. CNS:

1. Gait : Normal / Abnormal

2. Cranial nerves

(i) Olfactory: Normal/ Abnormal

(ii) Trigeminal: Normal/ Abnormal

(iii) Facial : Normal / Abnormal

(iv) Others : Normal / Abnormal

3. Motor system:

(i) Power

a. Muscles of hand : Normal / Abnormal

b. Muscles of foot : Normal / Abnormal

(ii) Tone : Normal / Abnormal

(iii) Reflexes : Normal / Abnormal

(iv) Coordination: Normal/ Abnormal

(v) Pen test : Positive / Negative

(vi) Book test : Positive / Negative

(vii)Card test : Positive / Negative

4. Sensory system:

(i) Fine touch: Normal / Impaired / Lost

(ii) Crude touch : Normal / Impaired / Lost

(iii) Pain : Normal / Impaired / Lost

(iv) Cold temperature : Normal / Impaired / Lost

(v) Warm temperature : Normal / Impaired / Lost

(vi) Pressure: Normal / Impaired / Lost

e. ANS

(i) Sweating : Normal/ Impaired / Lost

X. Dermatoscopic findings –

1. Yellow-orange areas	
2. Scales	
3.Xerosis	
4.Hair follicles	
5.Pigment network	
6.Vascular structures	
7.Follicles, sweat glands & appendages	
8.Other features	

XI. Histopathological findings -

1. Epidermis	
2. Subepidermal clear zone (Grenz zone)	
2. Dermis ● Epitheloid granuloma ● Macrophage granuloma ● Giant cells ● Lymphocytes ● Macrophages	

XII. Provisional Diagnosis

- **On Clinical Examination :**
- **On Dermoscopy :**
- **On Histopathology :**

XIII. Final Diagnosis :

XIV. Treatment Advised :

ANNEXURE III – PHOTOGRAPHS

Figure 1 a

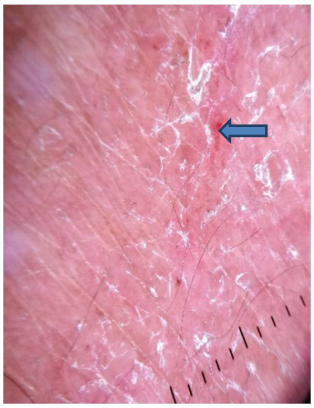


Figure 1 b - i

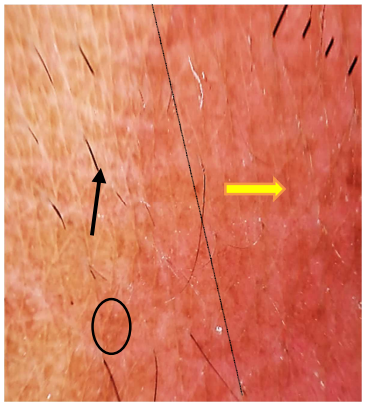


Figure 1b - ii

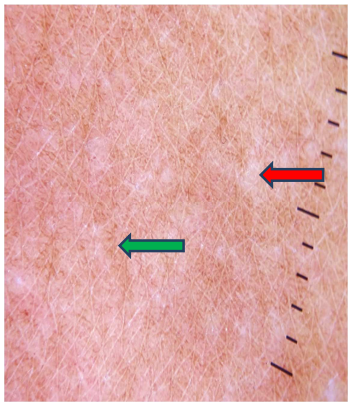


Figure 1b - iii

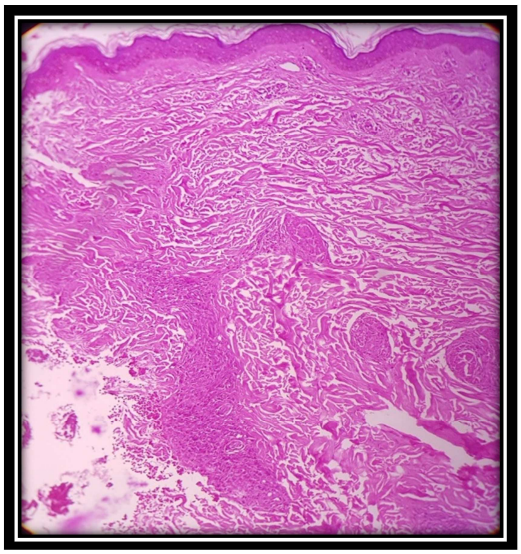


Figure 1 c – i

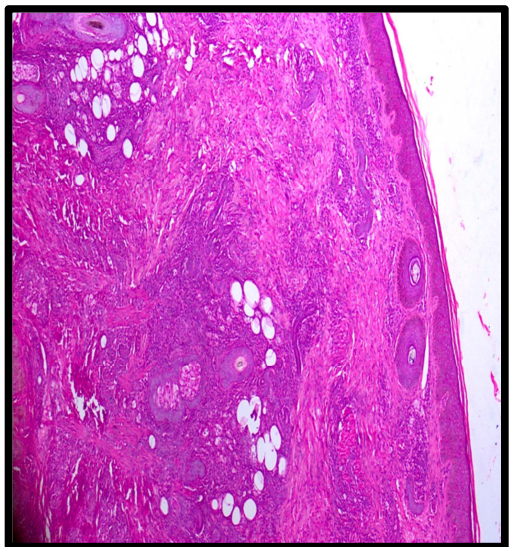


Figure 1 c – ii

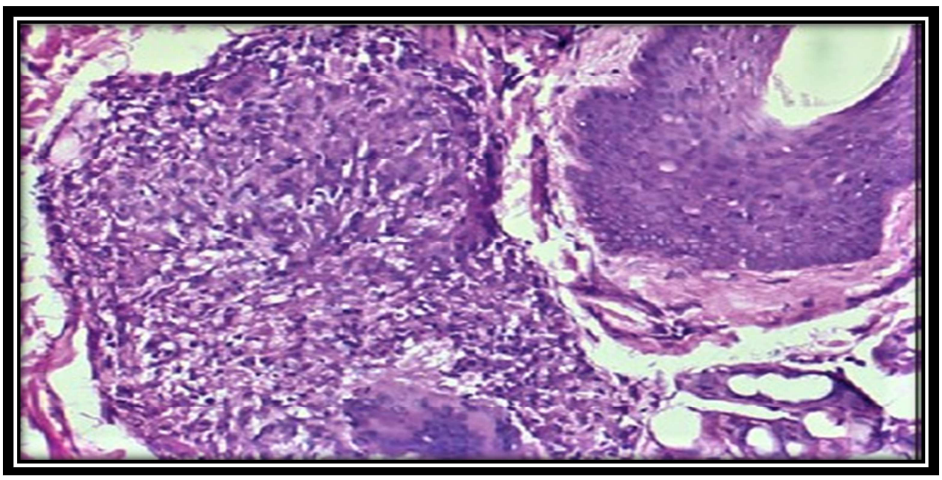


Figure 1 c – iii

Figure 1a-c: (a) Clinical image of borderline tuberculoid leprosy showing asymmetrical erythematous plaque with hypoesthesia and loss of hair with satellite lesions in its vicinity.(b- i, ii, iii) Dermoscopic image showing structureless yellow orange areas (yellow arrow), structureless white areas (red arrow), broken pigment network (green arrow), linear branching vessel (blue arrow), loss of hair follicles(black arrow) and white dots (black circle)in affected area (right to the line) as compared with normal skin (left to the line) (DermLite DL4,polarized contact,10x). (c-i,ii,iii) Histopathology showing numerous granulomas in the dermis and around the adnexal tissue, compromising of good number of lymphocytes (H&E ,40x,200x,400x).

Figure 2a

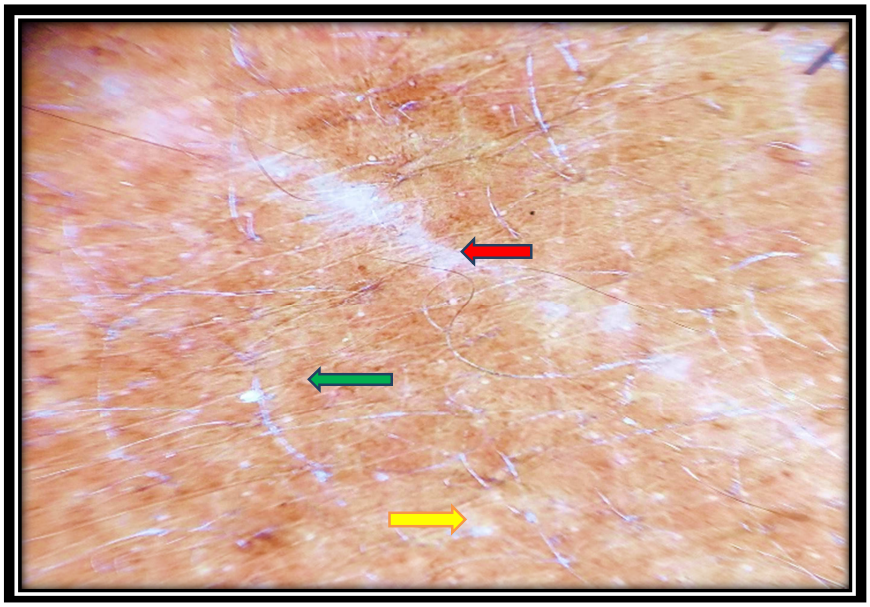


Figure 2b

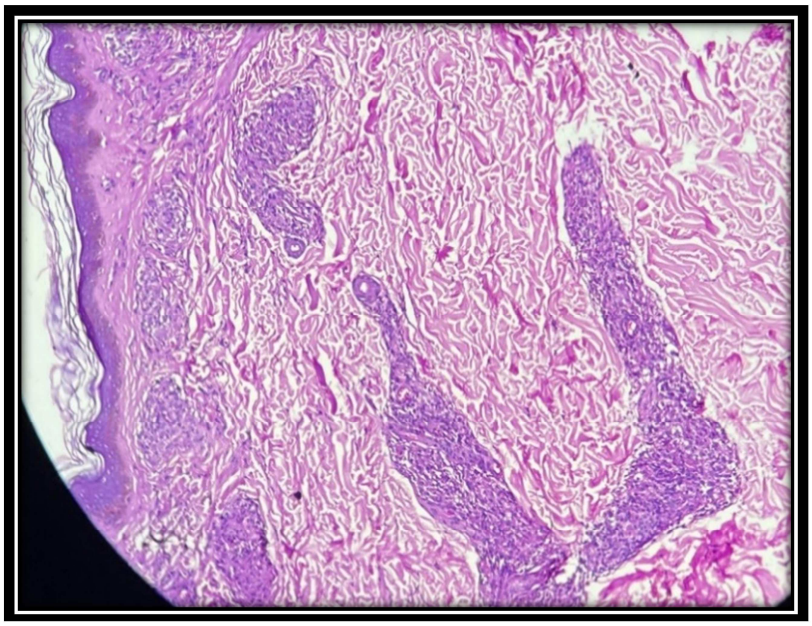


Figure 2c (i)

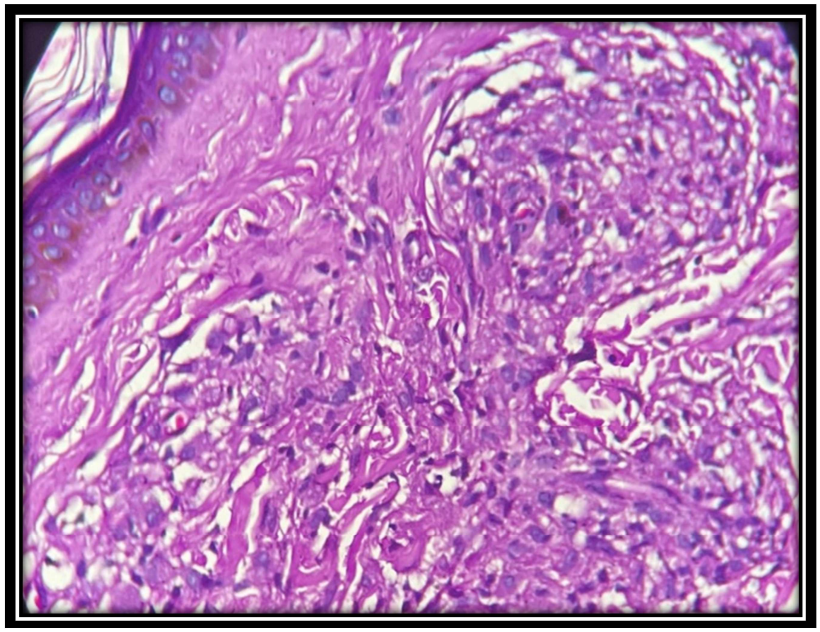


Figure 2c (ii)

Figure 2a-c: (a) Clinical image of borderline lepromatous leprosy showing symmetrical copper colored plaques with central elevation and peripheral sloping. (b) Dermoscopic image showing structureless yellow orange areas (yellow arrow), broken pigment network (green arrow), and characteristic linear white chrysalis areas (red arrow) (DermLite DL4, polarized contact,10x). (c-i ,ii) Histopathology showing grenz zone and ill defined aggregates of foamy cells admixed with epithelioid cells in the dermis. (H&E,200x, 400x).

Figure 3a – i

Figure 3a – ii

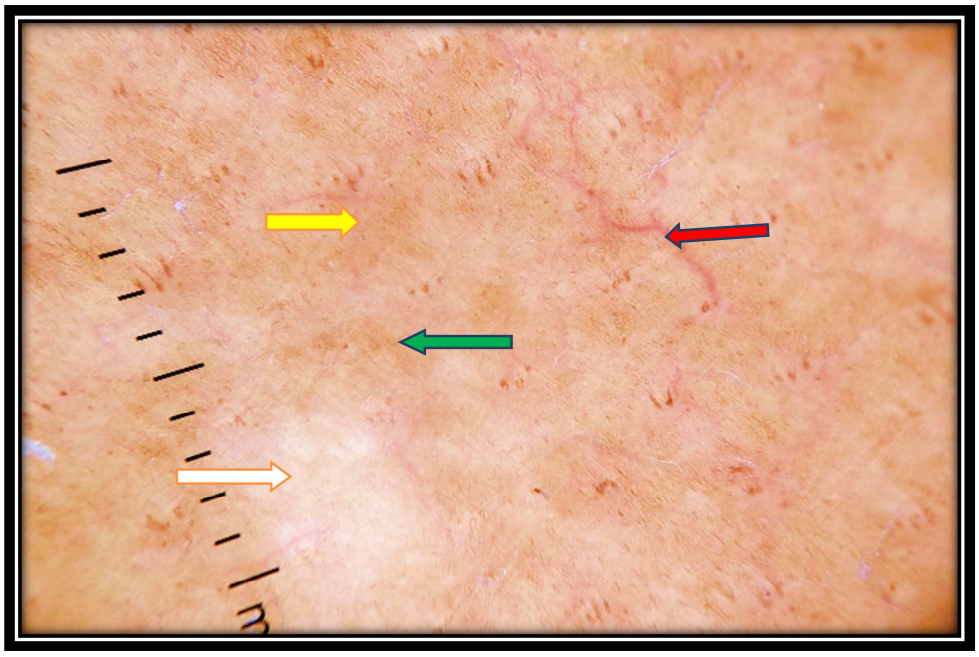


Figure 3b

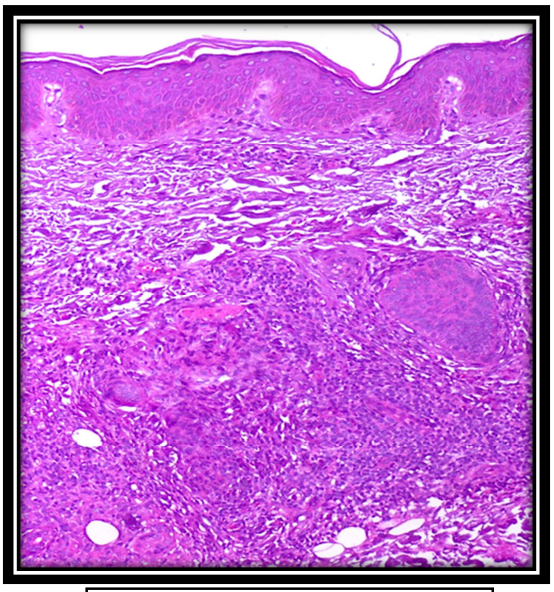


Figure 3c – i

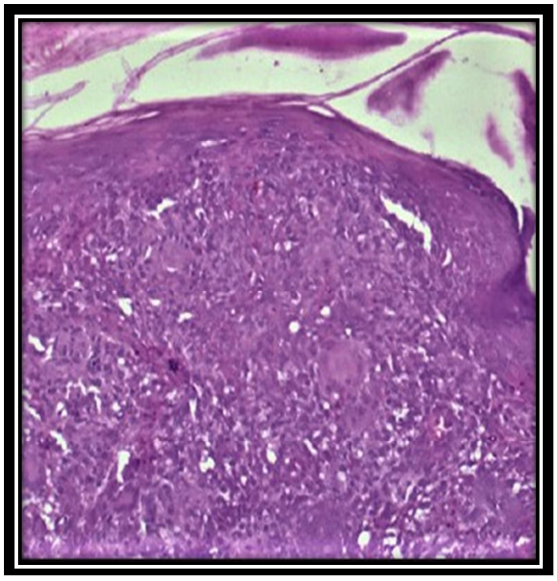


Figure 3c – ii

Figure 3a-c: (a) Clinical image of tuberculoid leprosy showing asymmetrical solitary anesthetic plaque with sharply defined and elevated border that slopes down to a flat atrophic centre (saucer right way up) with loss of hair follicles. (b) Dermoscopic image showing structureless yellow orange areas (yellow arrow), structureless white areas (white arrow), broken pigment network (green arrow), linear branching vessel (red arrow) and loss of hair follicle and white dots (DermLite DL4, polarized contact,10x). (c-i,ii) Histopathology showing granuloma with dense lymphocytic infiltrate in the dermis along with numerous Langhans giant cells, eroding the epidermis(H & E,100x,200x).

Figure 4a - i

Figure 4a - ii

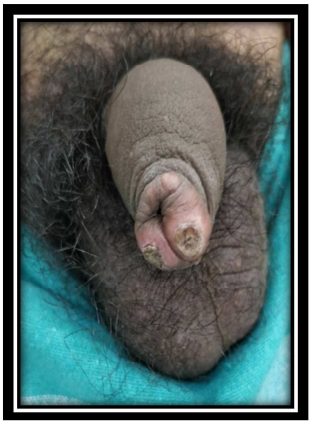


Figure 4a - iii

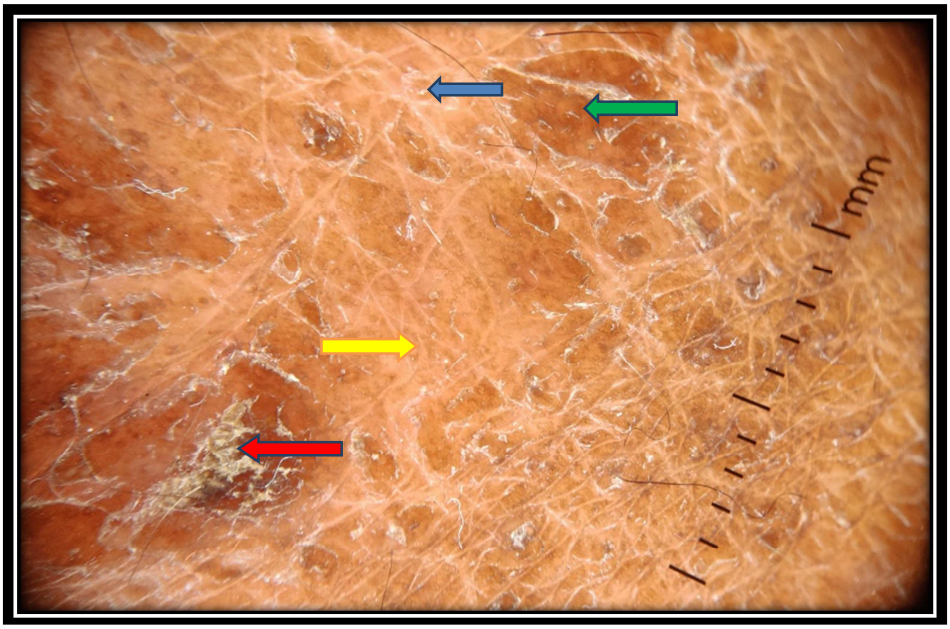


Figure 4b

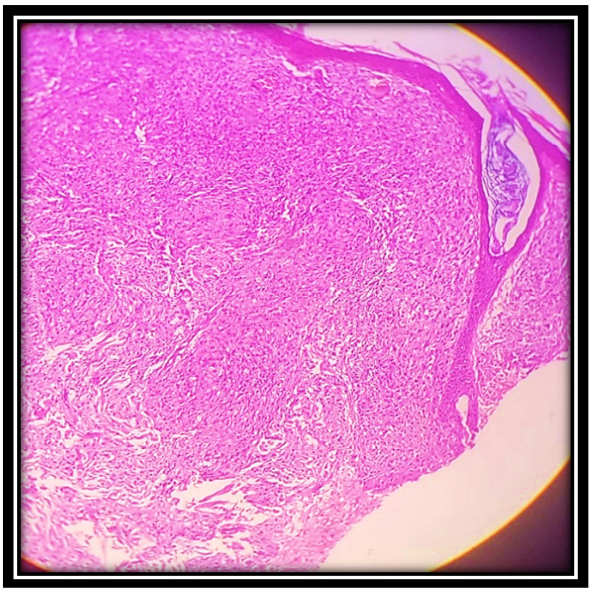


Figure 4c – i

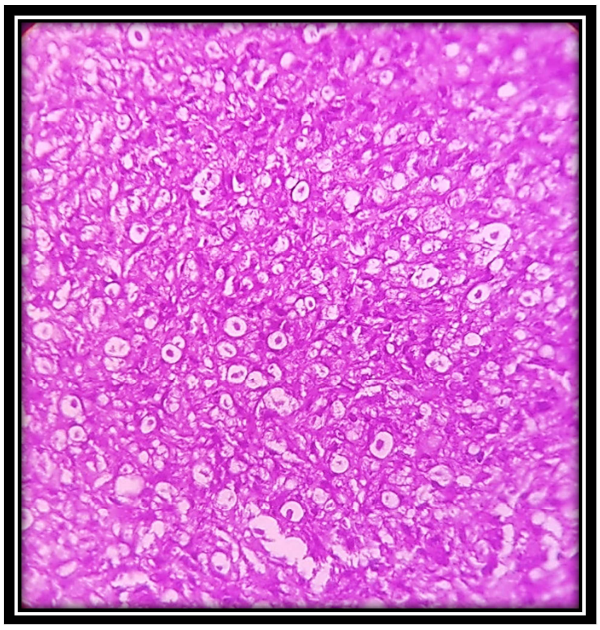


Figure 4c – ii

Figure 4a-c:(a) Clinical image of Lepromatous leprosy showing bilaterally symmetrical leproma nodules with xerotic skin and loss of hair follicles. (b) Dermoscopic image showing structureless yellow orange areas (yellow arrow) , accentuation of normal reticular pigment network (green arrow), loss of hair follicle (blue arrow) and characteristic white scaling (red arrow) (DermLite DL4, polarized contact, 10x).(c- i ,ii)Histopathology showing diffuse infiltration of the dermis by numerous foamy macrophages. (H & E ,40x,200x).

Figure 5a

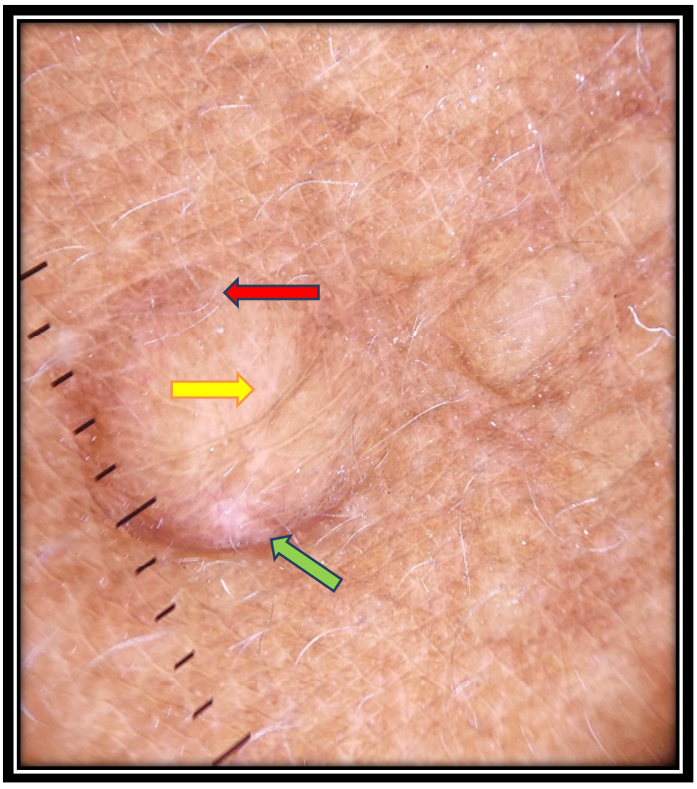


Figure 5b

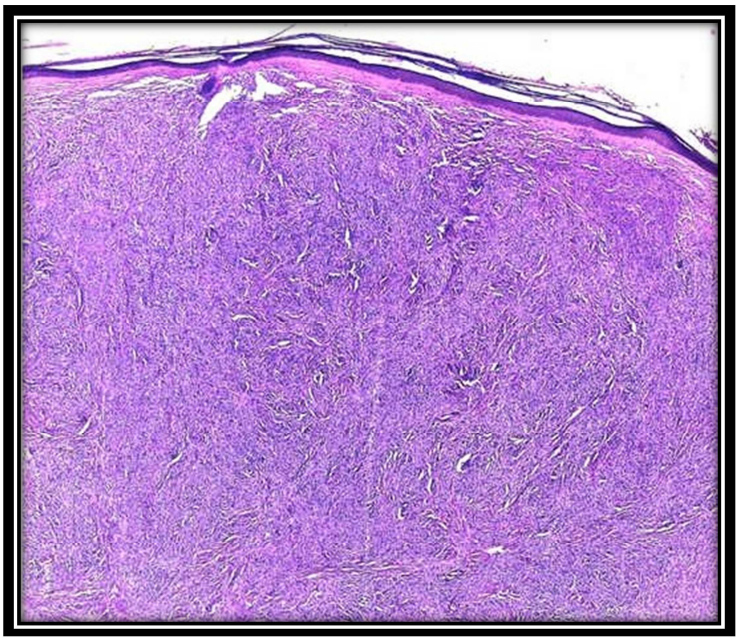


Figure 5c – i

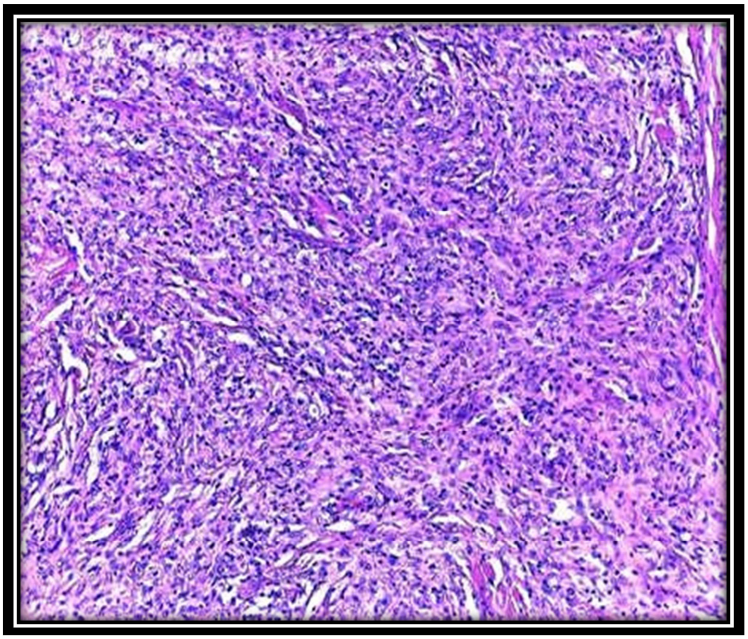


Figure 5c – ii

Figure 5a-c: (a) Clinical image of Histoid leprosy showing skin colored shiny, smooth nodules; (b)Dermoscopic image showing structureless yellow orange areas (yellow arrow), blanchable dome shaped structures (red arrow)with peripheral brown pigmentation (green arrow) (DermLite DL4, polarized contact,10x).(c- i ,ii) Histopathology showing expansile granuloma in dermis with spindle shaped cells (H &E, 40x, 100x).

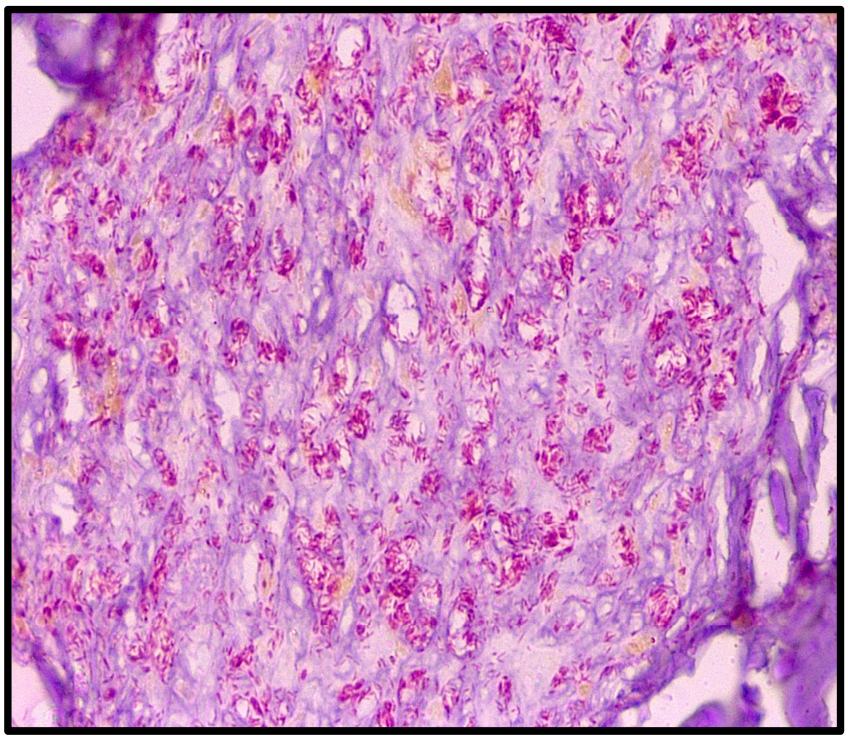


Figure 6 - Photomicrograph of lepromatous leprosy showing clusters of bacilli forming globi .BI +6 (Wade Fite stain ,200x)

ANNEXURE IV - KEY TO MASTER CHART

- Yes: Present
- No: Absent
- AFB – Acid fast bacilli

ANNEXURE V - MASTER CHART

								COMPLAINTS			
Serial no	Name	Age	Sex	Occupation	Socioeconomic Status	Rural/Urban area	Duration of symptoms(months)	Hypopigmented lesions with numbness	Erythematous raised lesion with numbness	Erythematous raised lesions without numbness	Skin colored raised lesions without numbness
1	Sachin Kamble	28	MALE	FARMER	LOW INCOME GROUP	RURAL	6	No	No	Yes	No
2	Veena Katti	39	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	6	Yes	No	No	No
3	Yallappa Badiger	35	MALE	FARMER	LOW INCOME GROUP	RURAL	12	No	No	Yes	No
4	Shashikant Mohite	55	MALE	CLERK	MODERATE INCOME GROUP	URBAN	20	No	No	Yes	No
5	Umesh Bhuvi	27	MALE	SHOPKEEPER	MODERATE INCOME GROUP	URBAN	8	No	No	Yes	No
6	Suvarna Emmi	36	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	12	No	No	Yes	No
7	Mahesh Kjenani	40	MALE	FARMER	LOW INCOME GROUP	RURAL	14	No	No	Yes	No
8	Shivaji Patil	42	MALE	FARMER	LOW INCOME GROUP	RURAL	7	No	No	Yes	No
9	Shivalila Mallapur	40	MALE	FARMER	LOW INCOME GROUP	RURAL	9	No	Yes	No	No
10	Laxman Mallikeri	40	MALE	FARMER	LOW INCOME GROUP	RURAL	15	No	Yes	No	No
11	Arjun Badakeri	39	MALE	LABOURER	LOW INCOME GROUP	URBAN	18	No	No	Yes	No
12	Siddappa Yallappa Hallur	55	MALE	CLERK	MODERATE INCOME GROUP	URBAN	24	No	Yes	No	No
13	Kalpana Bhaktande	45	FEMALE	HOUSEWIFE	LOW INCOME GROUP	RURAL	18	Yes	No	No	No
14	Savita Kamble	40	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	6	Yes	No	No	No
15	Yallawwa Nagral	36	FEMALE	FARMER	LOW INCOME GROUP	RURAL	8	No	Yes	No	No
16	Fakirappa Gugarnath	30	MALE	SHOPKEEPER	MODERATE INCOME GROUP	RURAL	12	No	Yes	No	No
17	Mariya Nadaf	19	FEMALE	STUDENT	MODERATE INCOME GROUP	URBAN	24	Yes	No	No	No
18	Adiveppa S.K.	38	MALE	FARMER	LOW INCOME GROUP	RURAL	24	No	Yes	No	No
19	Suvarna Mahadev Varagatte	35	FEMALE	FARMER	LOW INCOME GROUP	RURAL	22	No	Yes	No	No
20	Ritesh Navanath Balekundri	30	MALE	SHOPKEEPER	MODERATE INCOME GROUP	URBAN	20	Yes	No	No	No
21	Vijay Singh	47	MALE	SOLDIER	MODERATE INCOME GROUP	URBAN	14	No	No	No	Yes
22	Sanjay Kedari Auargal	48	MALE	FARMER	LOW INCOME GROUP	RURAL	12	No	Yes	No	No
23	Basavanni Medar	58	MALE	LABOURER	LOW INCOME GROUP	RURAL	6	No	No	Yes	No
24	Sanganna Tallur	41	MALE	SHOPKEEPER	MODERATE INCOME GROUP	URBAN	6	No	Yes	No	No
25	Neelawwa . M	45	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	6	No	No	Yes	No
26	Yashwant.D	48	MALE	LABOURER	LOW INCOME GROUP	RURAL	22	Yes	No	No	No
27	Mumtaj Ashiqali Anasari	38	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	24	No	No	No	Yes
28	Mohammad Rafiq Bagwan	44	MALE	SHOPKEEPER	MODERATE INCOME GROUP	URBAN	18	No	No	No	Yes
29	Mohammad Gouz Rizwan	50	MALE	FARMER	LOW INCOME GROUP	RURAL	20	No	Yes	No	No
30	Anandrao Patil	62	MALE	FARMER	LOW INCOME GROUP	RURAL	22	No	Yes	No	No
31	Basavaraj Kabbur	58	MALE	FARMER	LOW INCOME GROUP	RURAL	24	No	No	Yes	No
32	Prakash Kalloli	48	MALE	CLERK	MODERATE INCOME GROUP	URBAN	18	No	Yes	No	No
33	Sumitra Sanadi	39	FEMALE	HOUSEWIFE	LOW INCOME GROUP	RURAL	12	No	No	Yes	No
34	Dundappa Godi	58	MALE	FARMER	LOW INCOME GROUP	RURAL	12	Yes	No	No	No
35	Hanamantgouda Patil	62	MALE	LABOURER	LOW INCOME GROUP	RURAL	8	No	No	Yes	No
36	Manjula Waddar	45	FEMALE	HOUSEWIFE	LOW INCOME GROUP	RURAL	10	Yes	No	No	No
37	Shankar Dhanagar	52	MALE	FARMER	LOW INCOME GROUP	RURAL	24	No	Yes	No	No
38	Kaveri Kutabagi	48	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	20	No	Yes	No	No
39	Chanabasappa Rendale	59	MALE	FARMER	LOW INCOME GROUP	RURAL	18	No	No	Yes	No
40	Kalavati Kurbet	38	FEMALE	HOUSEWIFE	MODERATE INCOME GROUP	URBAN	12	No	No	Yes	No

	MORPHOLOGY OF SELECTED LESION			SITE OF SELECTED LESION				COLOR OF SELECTED LESION									CLINICAL DIAGNOSIS				
Serial no	Patch	Nodule	Plaque	Head & Neck	Upper Limb	Lower Limb	Trunk	Hypopigmented	Erythematous	Skin colored	Surface of lesion	Margins of lesion	Hypoesthesia	Hypotrichosis	Xerosis	Slit Skin Smear - AFB	Tuberculoid Leprosy	Borderline Tuberculoid	Borderline Lepromatous	Lepromatous Leprosy	Histoid Leprosy
1	No	No	Yes	No	No	No	Yes	No	Yes	No	Rough	Regular	No	No	Yes	Positive	No	No	Yes	No	No
2	Yes	No	No	No	No	No	Yes	Yes	No	No	Smooth	Irregular	Yes	Yes	No	Positive	No	Yes	No	No	No
3	No	Yes	No	No	Yes	No	No	No	Yes	No	Smooth	Regular	Yes	No	No	Positive	No	No	Yes	No	No
4	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Regular	No	No	No	Positive	No	No	Yes	No	No
5	No	Yes	No	No	No	No	Yes	No	Yes	No	Smooth	Regular	No	No	No	Positive	No	No	No	Yes	No
6	No	No	Yes	Yes	No	No	No	No	Yes	No	Smooth	Regular	Yes	Yes	No	Positive	No	Yes	No	No	No
7	No	No	Yes	No	No	Yes	No	No	Yes	No	Smooth	Regular	No	No	Yes	Negative	No	No	Yes	No	No
8	No	Yes	No	Yes	No	No	No	No	Yes	No	Smooth	Regular	No	No	No	Negative	No	No	Yes	No	No
9	No	No	Yes	No	No	No	No	No	Yes	No	Rough	Regular	Yes	Yes	Yes	Negative	Yes	No	No	No	No
10	No	No	Yes	No	Yes	No	Yes	No	Yes	No	Smooth	Regular	Yes	Yes	No	Negative	No	Yes	No	No	No
11	No	No	Yes	Yes	No	No	No	No	Yes	No	Smooth	Regular	No	No	Yes	Positive	No	No	Yes	No	No
12	No	No	Yes	No	Yes	No	No	No	Yes	No	Rough	Regular	Yes	Yes	No	Negative	No	Yes	No	No	No
13	Yes	No	No	No	No	Yes	No	Yes	No	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
14	Yes	No	No	No	Yes	No	No	Yes	No	No	Smooth	Irregular	Yes	No	No	Negative	No	Yes	No	No	No
15	No	No	Yes	No	No	Yes	No	No	Yes	No	Rough	Irregular	Yes	No	Yes	Negative	No	Yes	No	No	No
16	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
17	Yes	No	No	No	Yes	No	No	Yes	No	No	Smooth	Irregular	Yes	No	No	Negative	No	Yes	No	No	No
18	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Regular	Yes	Yes	No	Negative	Yes	No	No	No	No
19	No	No	Yes	No	Yes	No	No	No	Yes	No	Rough	Irregular	Yes	No	Yes	Negative	No	Yes	No	No	No
20	No	No	Yes	No	No	No	Yes	Yes	No	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
21	No	Yes	No	No	Yes	No	No	No	No	Yes	Smooth	Regular	No	No	No	Positive	No	No	No	No	Yes
22	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
23	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Irregular	No	No	Yes	Positive	No	No	No	Yes	No
24	No	No	Yes	No	No	Yes	No	No	Yes	No	Rough	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
25	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Irregular	No	No	Yes	Positive	No	No	Yes	No	No
26	No	No	Yes	No	Yes	No	Yes	Yes	No	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
27	No	Yes	No	No	Yes	No	No	No	No	Yes	Smooth	Regular	No	No	No	Positive	No	No	No	No	Yes
28	No	Yes	No	No	Yes	No	Yes	No	No	Yes	Smooth	Regular	No	No	No	Positive	No	No	No	No	Yes
29	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
30	No	No	Yes	No	No	No	Yes	No	Yes	No	Rough	Regular	No	No	Yes	Positive	No	No	Yes	No	No
31	No	No	Yes	No	Yes	No	Yes	No	Yes	No	Rough	Irregular	No	No	Yes	Positive	No	No	Yes	No	No
32	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Regular	Yes	Yes	No	Negative	Yes	No	No	No	No
33	No	Yes	No	No	No	Yes	No	No	Yes	No	Smooth	Irregular	No	No	No	Positive	No	No	No	Yes	No
34	Yes	No	No	No	No	No	Yes	Yes	No	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
35	No	Yes	No	No	No	No	Yes	No	Yes	No	Smooth	Irregular	No	No	No	Positive	No	No	No	Yes	No
36	Yes	No	No	No	Yes	No	No	Yes	No	No	Smooth	Irregular	Yes	Yes	No	Negative	No	Yes	No	No	No
37	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Regular	Yes	Yes	No	Negative	Yes	No	No	No	No
38	No	No	Yes	No	No	No	Yes	No	Yes	No	Smooth	Regular	Yes	Yes	No	Negative	Yes	No	No	No	No
39	No	No	Yes	No	No	Yes	No	No	Yes	No	Smooth	Regular	No	No	Yes	Negative	No	No	Yes	No	No
40	No	No	Yes	No	No	Yes	No	No	Yes	No	Smooth	Regular	No	No	Yes	Negative	No	No	Yes	No	No

	DERMATOSCOPIC FINDINGS									
Serial no	Structureless yellow orange areas	Structureless white areas	Broken pigment network	Accentuation of normal reticular pigment network	Linear white chrysalis areas	Decreased white dots (Eccrine glands)	Loss of Hair follicles	White scaling	Linear / Branching vessels	Blanchable dome shaped structures with peripheral brown hyperpigmentation
1	Yes	Yes	Yes	No	Yes	No	No	Yes	No	No
2	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	No
3	Yes	No	Yes	No	No	No	No	No	Yes	No
4	Yes	No	No	No	Yes	No	No	No	No	No
5	Yes	No	No	Yes	No	No	Yes	Yes	No	No
6	Yes	No	Yes	No	No	No	Yes	No	Yes	No
7	Yes	No	Yes	No	Yes	No	No	Yes	No	No
8	Yes	Yes	No	No	Yes	No	No	No	No	No
9	Yes	No	Yes	No	No	Yes	Yes	No	Yes	No
10	Yes	Yes	Yes	No	No	Yes	Yes	No	No	No
11	Yes	No	Yes	No	Yes	No	No	Yes	No	No
12	Yes	Yes	Yes	No	No	Yes	Yes	No	No	No
13	Yes	No	Yes	No	No	Yes	Yes	No	No	Yes
14	Yes	Yes	Yes	No	No	No	No	No	No	No
15	Yes	No	Yes	No	No	No	No	Yes	No	No
16	Yes	Yes	Yes	No	No	Yes	Yes	No	No	No
17	Yes	Yes	No	No	No	No	Yes	No	No	No
18	Yes	No	Yes	No	No	Yes	Yes	No	Yes	No
19	Yes	Yes	No	No	No	No	No	Yes	No	No
20	Yes	Yes	Yes	No	No	No	Yes	No	No	No
21	Yes	Yes	No	No	No	No	No	No	No	Yes
22	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	No
23	Yes	No	No	No	No	No	Yes	No	No	No
24	Yes	No	Yes	No	No	Yes	Yes	No	Yes	No
25	Yes	No	Yes	No	Yes	No	No	Yes	No	No
26	Yes	Yes	No	No	No	Yes	Yes	No	Yes	No
27	Yes	Yes	No	No	No	No	No	No	No	No
28	Yes	No	No	No	No	No	No	No	No	Yes
29	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	No
30	Yes	No	Yes	No	Yes	No	No	Yes	No	No
31	Yes	No	Yes	No	Yes	No	No	Yes	No	No
32	Yes	No	Yes	No	No	Yes	Yes	No	Yes	No
33	Yes	No	No	Yes	No	No	Yes	No	No	No
34	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	No
35	Yes	No	No	Yes	No	No	Yes	No	No	No
36	Yes	No	Yes	No	No	Yes	Yes	No	Yes	No
37	Yes	No	Yes	No	No	Yes	Yes	No	Yes	No
38	Yes	Yes	Yes	No	No	Yes	Yes	No	Yes	No
39	Yes	Yes	Yes	No	Yes	No	No	Yes	No	No
40	Yes	No	Yes	No	No	No	No	Yes	No	No

	HISTOPATHOLOGICAL DIAGNOSIS														
Serial no	Hyperkeratosis	Dermal Granuloma	Vascular dilatation in periphery of granuloma	Periappendageal granuloma	Involvement of Grenz zone and basal layer with damage to melanocytes	Perineural involvement by granuloma	Periadenexal inflammation	Foamy Macrophages in dermis	Giant cells	Expansile granuloma with spindle shaped cells	Tuberculoid leprosy	Borderline Tuberculoid	Borderline Lepromatous	Lepromatous leprosy	Histoid leprosy
1	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
2	No	Yes	Yes	Yes	Yes	No	No	No	Yes	No	No	Yes	No	No	No
3	No	Yes	Yes	No	No	No	No	No	No	Yes	No	No	No	No	Yes
4	No	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
5	Yes	Yes	No	No	No	No	Yes	Yes	No	No	No	No	No	Yes	No
6	No	Yes	Yes	Yes	Yes	Yes	No	No	Yes	No	No	Yes	No	No	No
7	Yes	Yes	No	No	Yes	No	No	Yes	No	No	No	No	Yes	No	No
8	No	Yes	No	No	Yes	No	No	Yes	No	No	No	No	Yes	No	No
9	No	Yes	Yes	Yes	Yes	Yes	No	No	No	No	Yes	No	No	No	No
10	No	Yes	No	No	No	Yes	No	No	Yes	No	No	Yes	No	No	No
11	Yes	Yes	No	No	Yes	No	No	Yes	No	No	No	No	Yes	No	No
12	No	Yes	No	Yes	No	No	No	No	No	No	No	Yes	No	No	No
13	No	Yes	No	Yes	No	No	No	No	No	No	No	Yes	No	No	No
14	No	Yes	No	No	No	No	No	No	No	No	No	Yes	No	No	No
15	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
16	No	Yes	Yes	Yes	Yes	No	No	No	No	No	No	Yes	No	No	No
17	No	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
18	No	Yes	Yes	Yes	Yes	No	No	No	Yes	No	Yes	No	No	No	No
19	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
20	No	Yes	No	Yes	No	No	No	No	No	No	No	Yes	No	No	No
21	No	Yes	No	No	No	No	No	No	No	Yes	No	No	No	No	Yes
22	No	Yes	Yes	No	Yes	No	No	No	Yes	No	No	Yes	No	No	No
23	No	Yes	No	No	No	No	Yes	Yes	No	No	No	No	No	Yes	No
24	No	Yes	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	No	No	No
25	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
26	No	Yes	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	No	No	No
27	No	Yes	No	No	Yes	No	No	Yes	No	No	No	No	No	Yes	No
28	No	Yes	No	No	No	No	No	No	No	Yes	No	No	No	No	Yes
29	No	Yes	Yes	Yes	No	No	No	No	Yes	No	No	Yes	No	No	No
30	Yes	Yes	No	No	Yes	No	No	Yes	No	No	No	No	Yes	No	No
31	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No	Yes	No	No
32	No	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No
33	No	Yes	No	No	No	No	Yes	Yes	No	No	No	No	No	Yes	No
34	No	Yes	Yes	Yes	No	No	No	No	Yes	No	No	Yes	No	No	No
35	No	Yes	No	No	No	No	Yes	Yes	No	No	No	No	No	Yes	No
36	No	Yes	Yes	Yes	No	No	No	No	Yes	No	No	Yes	No	No	No
37	No	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No
38	No	Yes	Yes	Yes	Yes	No	No	No	No	No	Yes	No	No	No	No
39	Yes	Yes	No	No	Yes	No	No	Yes	No	No	No	No	Yes	No	No
40	Yes	Yes	No	No	Yes	No	No	Yes	No	No	No	No	Yes	No	No