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Dermatoscopy In Hansen's Disease and Its Correlation with Clinical Spectrum and Histopathology: An Observational Study

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KEYWORDS

Hansen's disease, Dermoscopy, Clinicopathological correlation, Slit skin smear, Lepra reactions, Granulomatous dermatoses.

ABSTRACT:

Introduction: Hansen's disease is a chronic granulomatous infection of the skin and peripheral nerves with a broad clinicopathological spectrum, often leading to diagnostic delay due to its varied presentation. Dermoscopy is a non-invasive bedside tool that may assist in spectrum-based evaluation. The present study aimed to assess dermoscopic features across the clinical spectrum of Hansen's disease and correlate them with clinical and histopathological findings.

Methodology: An observational cross-sectional study was conducted over eighteen months in the Department of Dermatology, Integral Institute of Medical Sciences and Research, Lucknow. Seventy patients clinically diagnosed with Hansen's disease and supported by slit skin smear examination were included. Detailed clinical assessment, dermoscopic evaluation, slit skin smear analysis, and histopathological examination were performed, and findings were correlated across disease spectra.

Results: Most patients were aged twenty to forty-nine years, with male predominance and predominantly lower socioeconomic status. Hypoesthesia was the commonest presentation, with asymmetrical, plaque-predominant lesions and frequent surface changes. The ulnar nerve was most commonly involved. Borderline tuberculoid Hansen's disease was the predominant clinical, dermoscopic, and histopathological subtype. Yellow–orange areas were the most frequent dermoscopic finding, while AFB positivity and lymphocytic infiltrate predominated histologically. Dermoscopic findings showed a significant association with histopathology ($p = 0.004$; OR = 11.25).

Conclusion: Dermoscopy enhances diagnostic confidence in Hansen's disease by supporting spectrum-based assessment and improving clinico-histopathological correlation. Its routine integration may aid early diagnosis and guide biopsy decisions.

Introduction

Hansen's disease is a chronic infectious granulomatous disease caused by *Mycobacterium leprae*.[1] It is a slowly progressive disorder primarily affecting the skin and peripheral nerves, with varied clinicopathological forms determined by host immune status.[1] According to the World Health Organization, the disease is defined by hypopigmented or reddish skin lesions with definite sensory loss, peripheral nerve involvement, or a positive slit skin smear test for acid-fast bacilli.[2] The clinical spectrum is classified by the Ridley–Jopling system based on clinical, histopathological, bacteriological, and immunological parameters.[3] *M. leprae* grows optimally at temperatures between twenty-seven and thirty-three degrees Celsius, explaining its predilection for cooler body regions such as the skin, superficial nerves, and upper respiratory mucosa.[4] The organism also proliferates in nine-banded armadillos with lower core temperatures, found predominantly in the south-central United States.[5] Additional animal reservoirs include

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chimpanzees, mangabey monkeys, and cynomolgus macaques.[6] Genomic sequencing of *M. leprae* and *M. lepromatosis* demonstrates extensive pseudogenes and loss of key metabolic enzymes, supporting their obligate intracellular nature.[7] De novo sequencing suggests divergence from a common ancestor more than thirteen million years ago.[8] Leprosy reactions are acute immunological events superimposed on the chronic disease course. Type one reaction represents a delayed hypersensitivity response with inflammation of existing lesions and neural dysfunction.[9] Type two reaction, or erythema nodosum leprosum, manifests as tender erythematous papules or nodules with systemic features.[10] Cutaneous hyperpigmentation may occur secondary to clofazimine therapy.[11] Diagnosis is primarily clinical and supported by slit skin smear examination; however, confirmation often requires invasive techniques such as skin or nerve biopsy.[1] Dermoscopy is a non-invasive, in vivo bedside tool that allows visualization of subsurface skin structures up to the reticular dermis and is useful in evaluating granulomatous dermatoses.[12] While histopathology and slit skin smear remain the gold standard, identification of characteristic dermoscopic patterns can aid early diagnosis, minimize treatment delay, and sometimes obviate the need for biopsy.[13] Given the wide clinical variability of leprosy, often termed “the greatest imitator”.[11] and the rarity of histoid and indeterminate forms.[11] dermoscopy serves as a valuable adjunct. The present study aims to evaluate dermoscopic findings across the clinical spectrum of Hansen’s disease and correlate these features with clinical and histopathological findings, with emphasis on scaling patterns, pigmentation, vascular structures, and appendageal changes.

Material And Methods

The study was conducted in the Department of Dermatology, Integral Institute of Medical Sciences and Research, Lucknow. It was designed as an observational cross-sectional study carried out over a period of eighteen months. The study included seventy patients diagnosed clinically with Hansen’s disease and supported by slit skin smear examination. All patients newly diagnosed with Hansen’s disease and undergoing treatment for less than six months were included in the study. The study population comprised individuals aged between eighteen and sixty years. Patients with pure neuritic Hansen’s disease, those who did not provide informed consent, patients already on treatment for more than six months, and individuals below eighteen years or above sixty years of age were excluded from the study. A detailed history was obtained from each patient and recorded in a predesigned proforma. Information regarding demographic details such as age, sex, education, occupation, marital status, and place of residence was documented. Clinical history included the onset, progression, and duration of the disease. All patients underwent a thorough general physical examination, followed by a complete dermatological examination. Based on the clinical assessment, slit skin smear findings, and subsequent investigations, patients were evaluated further. The findings obtained from dermoscopic examination were later correlated with clinical and histopathological features across the various clinical spectra of Hansen’s disease. At the end of the study, a significant correlation was established between clinical, histopathological, and dermoscopic findings in patients with Hansen’s disease.

Statistical Analysis:

Statistical analysis was performed using SPSS software (SPSS Inc., Chicago, IL, USA) for Windows, version 26.0. Continuous variables were expressed as mean with standard deviation or as range, where appropriate. Dichotomous variables were presented as numbers and frequencies and were analysed using the Chi-square test. Comparison of means between two or more groups was performed using the Student’s t-test. A p-value of less than 0.05 or 0.001 was considered statistically significant.

Results

Most patients belonged to the economically productive age group of 20–49 years (72.86%), with a male predominance (64.3%) and a male-to-female ratio of 1.8:1. [Fig-1 and 2] Labourers (24.3%) and unemployed individuals (22.9%) formed the largest occupational groups. [Fig-3] The majority of patients belonged to lower socioeconomic classes (Class 3–5) (95.7%). [Fig-4] Hypoesthesia was the most common clinical feature (94.3%). [Fig-5] Disease duration was 1–2 years in 44.3% of patients, while 28.6% had symptoms for more than 3 years. Asymmetrical lesion distribution was noted in 64.3%, and multibacillary disease was suggested by >100 lesions in 28.6%. Sensory loss over lesions was present in 60.0%, while glove-and-stocking sensory loss was seen in 34.3%. [Table-1] Plaques were the most common lesions, while macules and patches were mainly hypopigmented. Erythema was limited to plaques, and shiny surface, hair loss, and decreased sweating were frequent surface changes. [Table-2] The ulnar nerve was the most frequently involved nerve, with thickening noted in up to 90.0%, followed by the common peroneal and radial cutaneous nerves. [Table-3] Slit skin smear positivity was seen in 47.1%, with BI grades ranging from 1+ to 5+. Borderline tuberculoid Hansen’s disease (BTHD) was the most common clinical variant. [Table-4] Borderline tuberculoid Hansen’s disease was the most common variant on both dermoscopy (25.7%) and histopathology

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(20.0%), followed by tuberculoid and lepromatous spectra. [Table-5] Yellow–orange areas were the predominant dermoscopic finding, with vascular and pigmentary alterations seen in about half of cases. Histopathology most frequently revealed AFB positivity and lymphocytic infiltrate, while other features were variably present. [Table-6] Both dermoscopic and histopathological features showed a strong and statistically significant association with clinical variants of Hansen's disease, with most features demonstrating distinct distribution patterns across the Ridley–Jopling spectrum. [Table-7] The association between dermoscopic and histopathological findings was statistically significant, as demonstrated by Fisher's exact test ($p = 0.004$). The odds ratio of 11.25 indicates that patients with positive dermoscopic findings were over eleven times more likely to have confirmatory histopathological features compared to those without dermoscopic findings. [Table-8]

Discussion

In the present study (N = 70), Hansen's disease predominantly affected the economically productive age group, with 72.86% of patients aged 20–49 years, most commonly 30–39 years (27.14%), comparable to Sinha R et al., [14], and Amala B et al., [15] A male predominance was observed (64.3%; M:F ≈ 1.8:1), similar to reports by Sinha R et al., [14], and Mohan N et al., [16] Socioeconomic vulnerability was prominent, with 95.7% belonging to lower socioeconomic classes (Class 3–5), including 47.1% from Class 5; labourers (24.3%) and unemployed individuals (22.9%) were most affected, consistent with observations by Semwal S et al., [17], and Banerjee K et al., [18] Hypoesthesia was the commonest clinical finding (94.3%), with reactional features noted in a subset, in agreement with Mohta A et al., [2] Delayed detection persisted, with 28.6% showing disease duration >3 years, alongside asymmetrical lesions (64.3%) and a significant multibacillary burden (>100 lesions in 28.6%), reflecting the broad spectrum reported in previous studies. [2,19] The present study demonstrated extensive neural involvement, with lesional sensory loss in 60%, glove-and-stocking anesthesia in 34.3%, motor deficits in 17.1%, and deformities such as claw hand in 17.1%, along with Grade 1 (18.6%) and Grade 2 disability (15.7%). Ulnar nerve thickening was most frequent (90.0% right; 84.3% left), followed by common peroneal and radial cutaneous nerve involvement. These findings align with observations by Mohan N et al., [16], who highlighted diagnostic challenges in intermediate subtypes leading to delayed treatment and deformity, and by Semwal S et al., [17], who emphasized the importance of clinicobacteriological and morphological correlation in preventing progressive neural damage. In the present study, slit-skin smear (SSS) positivity was observed in 47.1% of cases, with BI ranging from negative to 5+, indicating marked heterogeneity in bacillary load. TTHD cases were BI-negative, while borderline and lepromatous variants showed progressively higher BI grades, with BI 3+–5+ concentrated in BLHD, LLHD, histoid leprosy, and LLHD with type 2 lepra reaction. This bacteriological gradient correlated significantly with histopathological features such as AFB positivity, globi, foamy macrophages, grenz zone, and epidermal thinning ($p < 0.0001$). Similar inclusion of multibacillary and reactional cases has been reported by Mohta A et al., [2] and others. [17,19] The present study showed predominance of tuberculoid-end disease, with BTHD (25.7%) and TTHD (17.1%) together accounting for over 40% of cases, while BLHD, LLHD, histoid leprosy, and reactional variants were also well represented. Reactional states comprised 21.4% of patients, including BTHD with type 1 lepra reaction (10.0%) and LLHD with type 2 lepra reaction (11.4%). Reactional cases demonstrated significant associations with inflammatory dermoscopic features and characteristic histopathological findings such as neutrophilic infiltration, dermal edema, and higher BI grades ($p < 0.0001$). These observations are consistent with findings reported by Vinay K et al., [19] and others [2], underscoring the importance of identifying reactional states in spectrum-based assessment. In the present study, yellow–orange areas were the most frequent dermoscopic finding (78.57%), followed by linear vascular structures (51.43%), diminished pigment network (51.43%), decreased white dots (47.14%), loss of hair (44.29%), and structureless scaly areas and shiny white structures (47.14% each). Yellow–orange areas were significantly associated with borderline and lepromatous variants, including BTHD, BLHD, LLHD, histoid leprosy, and reactional states ($p = 0.0267$), while pigment network alterations, appendageal loss, and vascular patterns showed highly significant associations with BLHD, LLHD, histoid, and reactional variants ($p < 0.0001$). These findings are consistent with the granuloma signature described by Mohta A et al., [2] and vascular–granuloma correlations reported by Vinay K et al., [19] The present study emphasizes that the diagnostic value of dermoscopy in Hansen's disease lies in recognizing specific constellations of findings rather than isolated features. The combination of yellow–orange background coloration with loss of eccrine and follicular openings, appendageal loss, altered pigment network, and non-uniform vascular patterns was highly characteristic of leprosy. In contrast, other granulomatous disorders such as granuloma annulare, sarcoidosis, lupus vulgaris, necrobiosis lipoidica, and PKDL may show yellow–orange areas but typically preserve appendageal structures or demonstrate distinct vascular and surface features. In the present study, absent pigment network, decreased white dots, and loss of

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hair showed significant association with leprosy variants ($p < 0.0001$), findings uncommon in most mimickers. Hypopigmented dermatoses like vitiligo, pityriasis alba, and post-inflammatory hypopigmentation showed preserved appendageal openings, while psoriasis and tinea corporis demonstrated characteristic scaling and vascular patterns absent in leprosy. Overall, dermoscopy serves as a valuable adjunct in differentiating Hansen's disease when interpreted alongside clinical, neurological, bacteriological, and histopathological findings. The present study showed marked vascular heterogeneity in reactional states, with dotted vessels significantly associated with BLHD and LLHD with type 2 lepra reaction, and arborizing vessels exclusively seen in LLHD with type 2 lepra reaction ($p < 0.0001$). Increased erythema was present in all type 1 and type 2 reactions, indicating active inflammation. Histopathologically, neutrophilic infiltration and dermal edema predominated in LLHD with type 2 lepra reaction and BTHD with type 1 lepra reaction ($p < 0.0001$). These findings align with Errichetti E et al., [20] and Chopra A et al., [21], supporting dermoscopy in identifying inflammatory and vascular changes during lepra reactions. In the present study, histoid leprosy demonstrated distinctive dermoscopic features, with exclusive presence of crown vessels ($p < 0.0001$) and significant associations with shiny white structures, yellow globular structures, shiny surface, and absent pigment network. These findings closely correlated with histopathology, where spindle-shaped histiocytes were exclusively observed in histoid leprosy ($p < 0.0001$), along with high BI grades and foamy macrophages. This strong dermoscopy–histopathology concordance is consistent with reports by Acharya P et al., [16] and others. [2,19] Branching and honeycomb-shaped brownish hyperpigmentation was infrequent (2.86%) and showed no significant association with clinical variants ($p = 0.3699$). This contrasts with findings by Chopra A et al., [21] and Mohta A, et al., [2], possibly reflecting shorter treatment duration or timing of assessment. Nonetheless, its presence supports dermoscopy's ability to detect treatment-related pigmentary changes. The present study demonstrated clear spectrum-dependent histopathological patterns. Compact epithelioid granulomas, Langhans giant cells, epithelioid cells, and dense lymphocytic infiltrate were significantly associated with TTHD, BTHD, and BTHD with type 1 lepra reaction ($p < 0.0001$). Conversely, foamy macrophages, grenz zone, epidermal thinning, globi formation, and AFB positivity were significantly associated with BLHD, LLHD, histoid leprosy, and LLHD with type 2 lepra reaction ($p < 0.0001$). Spindle-shaped histiocytes were pathognomonic for histoid leprosy, while neutrophilic infiltration and dermal edema showed strong associations with reactional states, further strengthening clinico-pathological differentiation. Despite strong correlations, some cases showed non-specific histopathology, especially in early or borderline lesions. Similar discordance has been reported by Banerjee K, et al., [18] and others [16,17] attributing it to sampling limitations and immunological instability. A major strength of the present study is the significant association between dermoscopic positivity and histopathological confirmation (Fisher's exact $p = 0.004$; OR = 11.25). Dermoscopy-positive cases were over 11 times more likely to show confirmatory histopathology, especially with yellow–orange areas, appendageal loss, vascular changes, and pigment network alterations. These findings support reports by Vinay K et al., [19] and others. [2,21]

Conclusion

In conclusion, dermoscopy emerges as a practical, bedside, non-invasive modality that enhances diagnostic confidence, supports spectrum-based and reaction-oriented assessment, and guides biopsy decisions in Hansen's disease. The statistically significant associations demonstrated in the present study strongly support the integration of dermoscopy into routine diagnostic algorithms. However, the study was limited by its single-center design, modest sample size, and under-representation of certain subtypes, which may affect generalizability. Dermoscopic assessment was operator-dependent, and early or indeterminate lesions sometimes showed non-specific findings; treatment duration was also not uniformly analyzed, possibly influencing pigmentation patterns. Larger multicentric studies, standardized dermoscopic criteria, and longitudinal assessment of treatment-related changes are recommended, along with routine integration of dermoscopy and targeted training to improve diagnostic accuracy in Hansen's disease.

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TABLES AND FIGURES

TABLE-1: Clinical Profile of Patients with Hansen's Disease.

Parameter	Category	n (%)
Duration Of Illness	<1 year	19 (27.1)
	1–2 years	31 (44.3)
	>3 years	20 (28.6)
Arrangement	Asymmetrical	45 (64.3)
	Symmetrical	25 (35.7)
No. Of Skin Lesion	0–3	12 (17.1)
	4–10	25 (35.7)
	11–30	5 (7.1)
	31–100	8 (11.4)
	>100	20 (28.6)
Sensory Loss (Lesional)	Fine touch	42 (60.0)
	Temperature	42 (60.0)
	Pain	42 (60.0)
Sensory Loss (Glove & Stocking)	Fine touch	24 (34.3)
	Temperature	24 (34.3)
	Pain	24 (34.3)
Motor Examination	Motor deficit	12 (17.1)
	Claw hand	12 (17.1)
	Wrist drop	4 (5.7)
	Claw toes	5 (7.1)
	Foot drop	3 (4.3)
	Eye involvement	16 (22.9)
	Other deformities	16 (22.9)
Disability Grade	Disability Grade 0	46 (65.7)
	Disability Grade 1	13 (18.6)
	Disability Grade 2	11 (15.7)

TABLE-2: Morphological Characteristics and Surface–Appendageal Changes of Cutaneous Lesions in Hansen's Disease.

Characteristics	Feature	Present n (%)	Absent n (%)
Macule	Well-defined	10 (14.3)	60 (85.7)
	Regular margin	9 (12.9)	61 (87.1)
	Hypopigmented	10 (14.3)	60 (85.7)
	Erythematous	0	70 (100)
	Scaly	9 (12.9)	61 (87.1)
Patch	Well-defined	16 (22.9)	54 (77.1)
	Regular margin	7 (10.0)	63 (90.0)
	Hypopigmented	32 (45.7)	38 (54.3)
	Erythematous	0	70 (100)
	Scaly	5 (7.1)	65 (92.9)
Plaque	Well-defined	30 (42.9)	40 (57.1)
	Regular margin	20 (28.6)	50 (71.4)
	Raised border	23 (32.9)	46 (67.1)
	Hypopigmented	12 (17.1)	58 (82.9)
	Erythematous	16 (22.9)	54 (77.1)

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	Scaly	22 (31.4)	48 (68.6)
	Satellite Lesion	13(18.6)	57(81.4)
Surface & Appendageal Changes	Hair loss	37(52.9)	33(47.1)
	Decreased Sweating	37(52.9)	33(47.1)
	Shiny surface	40(57.1)	30(42.9)
	Skin infiltration	25 (35.7)	45 (64.3)
	Feeding nerve palpable	9 (12.9)	61 (87.1)
	Nodules	12(17.1)	58(82.9)

TABLE-3: Combined Peripheral Nerve Examination Findings.

Nerve	**Thickened**		**Tender**		**Nodules**	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**
Supraorbital	18 (25.7%)	15 (21.4%)	4 (5.7%)	4 (5.7%)	2 (2.9%)	2 (2.9%)
Infraorbital	3 (4.3%)	3 (4.3%)	2 (2.9%)	2 (2.9%)	2 (2.9%)	2 (2.9%)
Supraclavicular	7 (10.0%)	7 (10.0%)	4 (5.7%)	4 (5.7%)	0 (0%)	0 (0%)
Infraclavicular	6 (8.6%)	6 (8.6%)	4 (5.7%)	4 (5.7%)	0 (0%)	0 (0%)
Radial	23 (32.9%)	22 (31.4%)	4 (5.7%)	4 (5.7%)	0 (0%)	0 (0%)
Temporal	11 (15.7%)	9 (12.9%)	6 (8.6%)	6 (8.6%)	0 (0%)	0 (0%)
Ulnar	63 (90.0%)	59 (84.3%)	19 (27.1%)	19 (27.1%)	5 (7.1%)	7 (10.0%)
Median	12 (17.1%)	12 (17.1%)	4 (5.7%)	4 (5.7%)	0 (0%)	0 (0%)
Great auricular	23 (32.9%)	22 (31.4%)	5 (7.1%)	5 (7.1%)	0 (0%)	0 (0%)
Radial cutaneous	39 (55.7%)	22 (31.4%)	8 (11.4%)	6 (8.6%)	3 (4.3%)	3 (4.3%)
Common peroneal	37 (52.9%)	51 (72.9%)	16 (22.9%)	16 (22.9%)	5 (7.1%)	5 (7.1%)
Anterior tibial	20 (28.6%)	7 (10.0%)	15 (21.4%)	4 (5.7%)	0 (0%)	0 (0%)
Posterior tibial	23 (32.9%)	23 (32.9%)	15 (21.4%)	15 (21.4%)	0 (0%)	0 (0%)
Sural	5 (7.1%)	5 (7.1%)	2 (2.9%)	2 (2.9%)	0 (0%)	0 (0%)

TABLE-4: Bacteriological Profile and Clinical Spectrum of Hansen's Disease.

Parameter	**Category**	**n (%)**
Slit Skin Smear (SSS)	Positive	33 (47.1)
	Negative	37 (52.9)
Bacteriological Index (BI)	Negative	13 (18.6)
	0	13 (18.6)
	1+	9 (12.9)
	2+	8 (11.4)
	3+	11 (15.7)
	4+	10 (14.3)
	5+	6 (8.6)
Clinical variant (Ridley–Jopling)	TTHD	12 (17.1)
	BTHD	18 (25.7)

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	BTHD with T1LR	7 (10.0)
	BBHD	5 (7.1)
	BLHD	8 (11.4)
	LLHD	8 (11.4)
	LLHD with T2LR	8 (11.4)
	Histoid	4 (5.7)

TABLE-5: Dermoscopy and Histopathology Based Classification.

Variant	Dermoscopy	Histopathology
	n (%)	n (%)
TTHD	10 (14.3)	8 (11.4)
BTHD	18 (25.7)	14 (20.0)
BTHD with T1LR	7 (10.0)	7 (10.0)
BBHD	1 (1.4)	4 (5.7)
BLHD	7 (10.0)	7 (10.0)
LLHD	8 (11.4)	6 (8.6)
LLHD with T2LR	7 (10.0)	7 (10.0)
Histoid	4 (5.7)	4 (5.7)
Non-specific	8 (11.4)	13 (18.6)

TABLE-6: Dermoscopic and Histopathological Findings.

Findings		Present n (%)	Absent n (%)
Dermoscopic	Yellow-orange areas	55 (78.57%)	15 (21.43%)
	Diminished pigment network	36 (51.43%)	34 (48.57%)
	Decreased white dots	33 (47.14%)	37 (52.86%)
	Loss of hair	31 (44.29%)	39 (55.71%)
	Linear vascular structure	36 (51.43%)	34 (48.57%)
	Dotted vascular structure	22 (31.43%)	48 (68.57%)
	Arborizing vessels	7 (10.00%)	63 (90.00%)
	Structureless scaly areas	33 (47.14%)	37 (52.86%)
	Shiny white structures	33 (47.14%)	37 (52.86%)
	Increased erythema	18 (25.71%)	52 (74.29%)
	Yellow globular structures	31 (44.29%)	39 (55.71%)
	Absent pigment network	26 (37.14%)	44 (62.86%)
	Shiny surface	19 (27.14%)	49 (70.00%)
	Follicular plugging	22 (31.43%)	46 (65.71%)
	Crown vessels	4 (5.71%)	64 (91.43%)
	Branching & honeycomb-shaped brownish hyperpigmentation	2 (2.86%)	68 (97.14%)
Histopathological	Compact epithelioid granuloma	25 (35.7)	45 (64.3)
	Langhans giant cells	29 (41.4)	41 (58.6)
	Foamy macrophages	24 (34.3)	46 (65.7)
	Lymphocytic infiltrate	36 (51.4)	34 (48.6)
	Grenz zone	28 (40.0)	42 (60.0)
	Epidermal thinning	24 (34.3)	46 (65.7)
	AFB positive	44 (62.9)	26 (37.1)
	Globi formation	21 (30.0)	49 (70.0)
	Epithelioid cells	33 (47.1)	37 (52.9)

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	Spindle-shaped histiocytes	4 (5.7)	66 (94.3)
	Neutrophilic infiltration	10 (14.3)	60 (85.7)
	Destruction of adnexa	35 (50.0)	35 (50.0)
	Dermal edema	18 (25.7)	52 (74.3)

TABLE-7: Association of Dermoscopic and Histopathological Findings with Clinical Variants of Hansen's Disease.

Findings		TTHD n(%)	BTHD n(%)	BBHD n(%)	BLHD n(%)	LLHD n(%)	HISTOID n(%)	BTHD-T1LR n(%)	LLHD-T2LR n(%)	χ^2 value	p-value
Dermoscopic feature	Yellow-orange areas	8 (66.7)	16 (88.9)	1 (20)	6 (75)	6 (75)	4 (100)	7 (100)	8 (100)	16.884	0.0267*
	Diminished pigment network	10 (83.3)	18 (100)	1 (20)	0 (0)	0 (0)	0 (0)	7 (100)	0 (0)	49.62	<0.00001*
	Decreased white dots	10 (83.3)	12 (66.7)	0 (0)	0 (0)	0 (0)	0 (0)	7 (100)	4 (50)	46.91	<0.00001*
	Loss of hair	7 (58.3)	13 (72.2)	0 (0)	0 (0)	0 (0)	0 (0)	7 (100)	4 (50)	42.777	<0.00001*
	Linear vascular structures	10 (83.3)	18 (100)	2 (20)	0 (0)	0 (0)	0 (0)	10 (100)	0 (0)	51.03	<0.00001*
	Dotted vascular structures	0 (0)	0 (0)	0 (0)	7 (87.5)	8 (100)	0 (0)	0 (0)	7 (87.5)	57.888	<0.00001*
	Arborizing vessels	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	7 (87.5)	61.24	<0.00001*
	Structureless scaly areas	7 (58.3)	18 (100)	1 (20)	0 (0)	0 (0)	0 (0)	7 (100)	0 (0)	50.366	<0.00001*
	Shiny white structures	0 (0)	0 (0)	1 (20)	6 (75)	8 (100)	4 (100)	7 (100)	7 (87.5)	58.411	<0.00001*
	Increased erythema	2 (16.7)	2 (11.1)	0 (0)	0 (0)	0 (0)	0 (0)	7 (100)	7 (87.5)	54.112	<0.00001*
	Yellow globules	0 (0)	0 (0)	0 (0)	5 (62.5)	8 (100)	4 (100)	7 (100)	7 (87.5)	60.009	<0.00001*
	Absent pigment network	0 (0)	0 (0)	0 (0)	7 (87.5)	8 (100)	4 (100)	0 (0)	7 (87.5)	59.444	<0.00001*
	Branching & honeycomb hyperpigme	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (14.3)	1 (12.5)	7.34	0.3699

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	ntation										
	Shiny surface	0 (0)	0 (0)	0 (0)	7 (87.5)	8 (100)	4 (100)	0 (0)	0 (0)	6.84	<0.00001*
	Follicular plugging	0 (0)	0 (0)	0 (0)	7 (87.5)	8 (100)	0 (0)	7 (100)	0 (0)	4.58	<0.00001*
	Crown vessel	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	4 (100)	0 (0)	0 (0)	8.95	<0.00001*
Histopathological feature	Compact epithelioid granuloma	8 (66.7)	10 (55.6)	0 (0)	0 (0)	0 (0)	0 (0)	7 (100)	0 (0)	48.92	<0.00001*
	Langhans giant cells	8 (66.7)	10 (55.6)	1 (20)	3 (37.5)	0 (0)	0 (0)	7 (100)	0 (0)	31.64	0.0001*
	Foamy macrophages	0 (0)	0 (0)	3 (60)	4 (50)	6 (75)	4 (100)	0 (0)	7 (87.5)	56.71	<0.00001*
	Lymphocytic infiltrate	8 (66.7)	14 (77.8)	3 (60)	4 (50)	0 (0)	0 (0)	7 (100)	0 (0)	39.18	<0.00001*
	Grenz zone	0 (0)	4 (22.2)	3 (60)	4 (50)	6 (75)	4 (100)	0 (0)	7 (87.5)	52.444	<0.00001*
	Epidermal thinning	0 (0)	0 (0)	8 (80)	7 (87.5)	7 (75)	10 (100)	0 (0)	3 (37.5)	06	01*
	AFB positivity	0 (0)	10 (55.6)	3 (60)	7 (87.5)	6 (75)	4 (100)	7 (100)	7 (87.5)	61.83	<0.00001*
	Globi formation	0 (0)	4 (22.2)	1 (20)	7 (87.5)	6 (75)	0 (0)	0 (0)	3 (37.5)	45.777	<0.00001*
	Epithelioid cells	8 (66.7)	10 (55.6)	3 (60)	5 (62.5)	0 (0)	0 (0)	7 (100)	0 (0)	41.29	<0.00001*
	Spindle-shaped histiocytes	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	4 (100)	0 (0)	0 (0)	28.16	<0.00001*
	Neutrophilic infiltration	3 (25)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	7 (87.5)	46.38	<0.00001*
	Destruction of adnexa	8 (66.7)	12 (66.7)	1 (20)	3 (37.5)	0 (0)	0 (0)	7 (100)	4 (50)	33.02	0.0009*
	Dermal edema	0 (0)	0 (0)	4 (80)	0 (0)	0 (0)	0 (0)	7 (100)	7 (87.5)	52.889	<0.00001*

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TABLE-8: Clinical Findings vs Histopathological Findings.

Dermoscopic Category		Histopathological Findings			Fisher's Exact Test
		Present n (%)	Absent n (%)	Total n (%)	
Dermoscopy Findings	Present (n=62)	54 (77.14 %)	8 (11.43 %)	62 (88.57 %)	p=0.004* OR=11.25
	Absent (n=8)	3 (4.29 %)	5 (7.14 %)	8 (11.43 %)	
	Total (N = 70)	57 (81.43 %)	13 (18.57 %)	70 (100 %)	

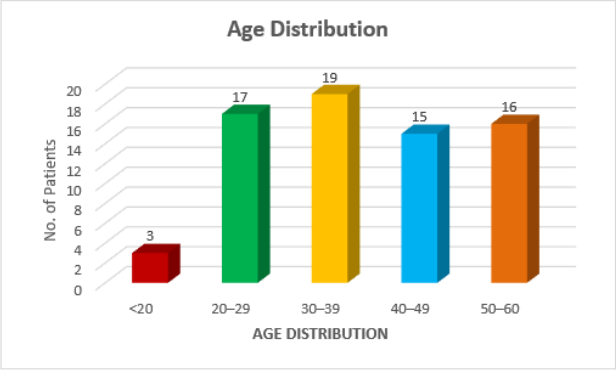

FIGURE-1: Age Distribution of Study Participants.

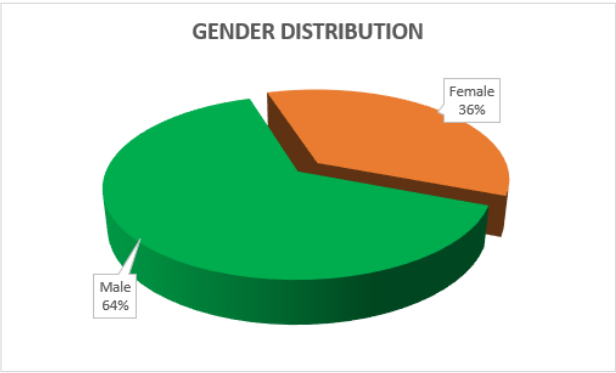

FIGURE-2: Gender Distribution of Study Participants.

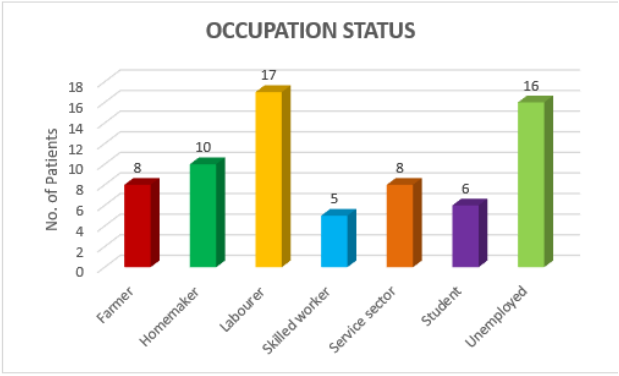

FIGURE-3: Occupational Status of Study Participants.

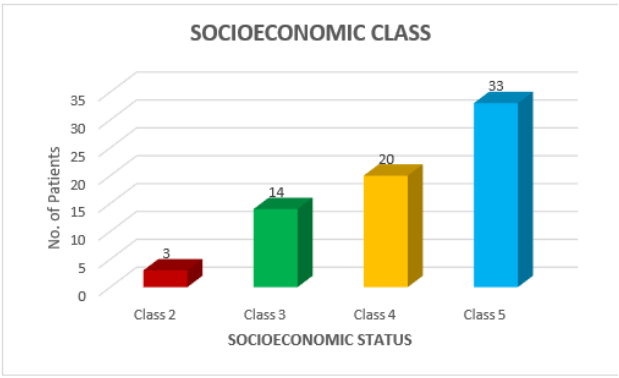

FIGURE-4: Socioeconomic Status of Study Participants.

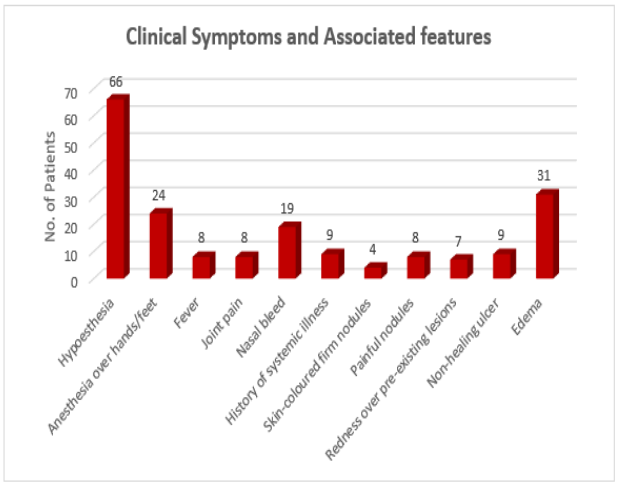

FIGURE-5: Clinical Symptoms and Associated Features.

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REFERENCES

1. Shreya K, Panwar H, Asati DP, Lakshman AM, Verma P, Denla Y. A Single Centre Cross-Sectional Observational Study on Dermatoscopy of Oral Mucosal Disorders and Histopathological Correlation in Tertiary Care Centre from Central India. Indian Journal of Dermatology. 2025 Jul 1;70(4):177-87.
2. Mohta A, Jain SK, Agrawal A, Kushwaha RK, Sharma P, Sethia K, Jain M. Dermoscopy in leprosy: a clinical and histopathological correlation study. Dermatology practical & conceptual. 2021 Apr 12;11(2):e2021032.
3. Ridéey DS, Jopling WH. Classification of leprosy according to immunity. A five-group system.
4. Scollard DM, Adams LB, Gillis TP, Krahenbuhl JL, Truman RW, Williams D. The continuing challenges of leprosy. Clinical microbiology reviews. 2006 Apr;19(2):338-81.
5. Truman R. Leprosy in wild armadillos. Leprosy review. 2005 Sep 1;76(3):198-208.
6. Valverde CR, Canfield D, Tarara R, Esteves MI, Gormus BJ. Spontaneous leprosy in a wild-caught cynomolgus macaque. International journal of leprosy and other mycobacterial diseases. 1998 Jun 1;66(2):140-8.
7. Cole ST, Eiglmeier K, Parkhill J, James KD, Thomson NR, Wheeler PR, Honore N, Garnier T, Churcher C, Harris D, Mungall K. Massive gene decay in the leprosy bacillus. Nature. 2001 Feb 22;409(6823):1007-11.
8. Singh P, Benjak A, Schuenemann VJ, Herbig A, Avanzi C, Busso P, Nieselt K, Krause J, Vera-Cabrera L, Cole ST. Insight into the evolution and origin of leprosy bacilli from the genome sequence of Mycobacterium lepromatosis. Proceedings of the National Academy of Sciences. 2015 Apr 7;112(14):4459-64.
9. Jopling's Handbook of leprosy (Page no:8,9) (7th Edition).Edited by Kabir Sardana and Ananta Khurana.
10. Lienhardt C, Fine PE. Type 1 reaction, neuritis and disability in leprosy. What is the current epidemiological situation?. Leprosy Review. 1994 Mar 1;65(1):9-33.
11. Parida SK, Grau GE, Zaheer SA, Mukherjee R. Serum tumor necrosis factor and interleukin 1 in leprosy and during lepra reactions. Clinical immunology and immunopathology. 1992 Apr 1;63(1):23-7.
12. Sonthalia S, Pasquali P, Agrawal M, Sharma P, Jha AK, Errichetti E, Lallas A, Sehgal VN. Dermoscopy update: Review of its extradiagnostic and expanding indications and future prospects. Dermatology practical & conceptual. 2019 Oct 31;9(4):253.
13. Acharya P, Mathur MC. Clinicodermoscopic study of histoid leprosy: a case series. International Journal of Dermatology. 2020 Mar;59(3):365-8.
14. Sinha R, Kumari P, Pallavi UK, Kumar B. A Clinical study of Hansen's Disease and Histopathological Correlation from a Tertiary Care Centre in East India. International Archives of Integrated Medicine. 2023 Feb 1;10(2):28-34.
15. Amala B, Balaji A, Kumar PB, et al. A clinicopathological correlation study of skin lesions in leprosy: An experience in a tertiary care hospital in South India. J Lab Physicians. 2022;14(1):15-20.
16. Mohan N, Mishra N. Clinico histopathological correlation within the spectrum of Hansen's disease: A multicentric study in North India. International Journal of Medical Research & Health Sciences. 2013;2(4):887-92.
17. Semwal S, Joshi D, Goel G, Asati D, Kapoor N. Clinico-histological correlation in Hansen's disease: Three-year experience at a newly established tertiary care center in central India. Indian journal of dermatology. 2018 Nov 1;63(6):465-8.
18. Banerjee K, Srikanth S. Clinicopathological correlation of Hansen's disease–A study of 75 cases. Scholars Journal of Applied Medical Sciences. 2016;4(12A):4249-53.
19. Vinay K, Kamat D, Chatterjee D, Narang T, Dogra S. Dermatoscopy in leprosy and its correlation with clinical spectrum and histopathology: a prospective observational study. Journal of the European Academy of Dermatology and Venereology. 2019 Oct;33(10):1947-51.
20. Errichetti E, Ankad BS, Jha AK, Sonthalia S, Akay BN, Bakos R, Bhat YJ, Bosseila M, Braun R, Cabo H, Cohen Sabban EN. International Dermoscopy Society criteria for non-neoplastic dermatoses (general dermatology): validation for skin of color through a Delphi expert consensus. International Journal of Dermatology.2021;61(4):461-471.
21. Chopra A, Mitra D, Agarwal R, Saraswat N, Talukdar K, Solanki A. Correlation of dermoscopic and histopathologic patterns in leprosy–a pilot study. Indian Dermatology Online Journal. 2019 Nov 1;10(6):663-8.