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Dermatoscopy in Hansen's Disease and Its Correlation with Clinical Spectrum and Histopathology: A Narrative Review

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KEYWORDS

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ABSTRACT:

Background- Hansen's disease (leprosy) is a chronic granulomatous infection caused by Mycobacterium leprae that primarily affects the skin and peripheral nerves. Its clinical and histopathological spectrum depends on host immune response and often overlaps with other inflammatory and granulomatous dermatoses, contributing to diagnostic delay. To summarize and critically synthesize evidence on dermatoscopy in leprosy, with emphasis on dermatoscopic patterns across the disease spectrum and their correlation with clinical morphology, bacillary burden, reactional states, and histopathology.

Methods- Narrative review of relevant literature describing dermatoscopic findings in Hansen's disease, including studies evaluating spectrum-wise patterns, reactional states, and clinico-histopathological correlations, along with supporting literature on dermatoscopy principles and granulomatous differentials.

Results- Conventional diagnosis relies on clinical evaluation supported by slit skin smear and histopathology, but these methods can be invasive and limited by sampling and interpretive variability. Dermatoscopy provides a non-invasive, repeatable bedside tool that improves recognition of leprosy-associated patterns. Frequently reported dermatoscopic features include yellow–orange structureless areas, focal white areas/white shiny structures, pigment network disruption, appendageal alterations, and characteristic vascular patterns (e.g., linear/branching vessels; crown vessels in histoid leprosy). These findings correlate with granulomatous infiltrates, fibrosis/collagen alteration, vascular displacement, and adnexal involvement on histopathology, and may aid recognition of reactional activity through increased erythema, vascular prominence, scaling, and pattern shift. Overlap exists with other granulomatous dermatoses; therefore, dermatoscopy is best interpreted in clinical context and used to guide targeted biopsy.

Conclusion- Dermatoscopy is a valuable adjunct in Hansen's disease that can strengthen bedside suspicion, support spectrum-wise assessment, improve biopsy site selection, and enhance clinico-histopathological correlation. Standardized terminology and larger spectrum-stratified diagnostic accuracy studies are needed to define reproducible criteria and clarify performance against granulomatous mimickers.

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Introduction

Hansen's disease remains a significant cause of preventable neuropathy and disability in endemic regions and continues to pose diagnostic challenges due to its wide clinical variability and overlap with common dermatoses. The disease primarily affects the skin and peripheral nerves, and delayed recognition can lead to permanent sensory loss, deformity, and social stigma. Comprehensive reviews emphasize that despite effective multidrug therapy, the continuing challenges of leprosy include delayed diagnosis, reactions, and disability prevention needs. [1]

A central concept in leprosy is that clinicopathological manifestations represent a continuous immunological spectrum. The Ridley–Jopling system classifies leprosy according to immunity, integrating clinical presentation, histology, and bacillary burden, and remains the most informative framework for understanding the spectrum. [2] For programmatic treatment decisions, simpler classification approaches have been proposed and used, particularly in settings where full laboratory support is limited. [3]

From a public health perspective, WHO reports document continued new case detection despite major reductions in prevalence, highlighting the need for improved early case recognition and supportive diagnostic tools. [4,5] In addition, leprosy is not exclusively confined to traditional endemic zones; zoonotic transmission has been supported in the southern United States, reinforcing the importance of maintaining diagnostic suspicion in appropriate clinical contexts. [6]

Because clinical examination alone may be insufficient—especially in subtle, early, or atypical lesions—there is increasing interest in non-invasive bedside adjuncts. Dermatoscopy has expanded from tumor diagnostics to inflammatory and infectious dermatoses, including granulomatous disorders. This review focuses on dermatoscopy in Hansen's disease and its correlation with clinical spectrum and histopathology, presenting a corrected, internally consistent manuscript with continuous in-text reference numbering.

Methods

This is a narrative review synthesizing key peer-reviewed literature relevant to: (a) leprosy biology, classification, reactions, and histopathology; (b) dermatoscopy principles; and (c) studies describing dermatoscopic patterns in leprosy and their clinicopathological correlations, including prospective, multicentre, and clinicopathological correlation studies. Evidence on dermatoscopic interpretation of granulomatous dermatoses was also used to contextualize overlaps and differentials.

Epidemiology And Public Health Context

WHO operational reports have documented that, although global prevalence has declined substantially, leprosy persists with ongoing new case detection, particularly in endemic countries, and programmatic emphasis remains on early diagnosis, timely treatment, and disability prevention. [4,5] A critical distinction is that WHO "elimination as a public health problem" (prevalence below a defined threshold) does not equate to eradication; continued transmission and late detection remain key concerns.

Leprosy epidemiology also includes zoonotic considerations. A landmark study reported probable zoonotic leprosy in the southern United States, linking human cases to armadillo exposure and supporting non-human reservoirs as relevant contributors to transmission in specific settings. [6] Complementing this, work specifically describing leprosy in wild armadillos highlights their role as a natural reservoir and a useful model for studying infection dynamics. [7] These findings underscore the broader geographic relevance of leprosy and the importance of tools that support early recognition across diverse clinical settings.

MICROBIOLOGY, GENOMICS, AND PATHOGENESIS

Genomic constraints and chronicity

M. leprae has a uniquely reduced genome with extensive gene decay and a large number of pseudogenes, which helps explain its slow growth, strict host dependence, and chronic disease course. The demonstration of massive gene decay in M. leprae has been central in understanding its obligate intracellular behavior. [8] Sequencing and evolutionary analysis of related leprosy bacilli has further informed hypotheses about divergence and origin, offering deeper insight into the phylogeny of leprosy pathogens. [9]

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Host response and tissue tropism

Leprosy pathophysiology is strongly shaped by host immune response, which determines clinical subtype, bacterial burden, and histopathology. An overview of pathophysiology emphasizes the interplay between bacillary persistence, granulomatous inflammation, and peripheral nerve involvement. [10] Foundational immunopathological discussions describe how the spectrum reflects differences in effective cell-mediated immunity versus ineffective containment and high bacillary loads. [11]

A key clinical implication of this biology is that the same pathogen can produce markedly different skin morphologies and histological patterns, making a purely morphology-based diagnosis unreliable in many contexts—particularly in early or borderline disease.

CLINICAL SPECTRUM AND CLASSIFICATION

Ridley–Jopling classification

The Ridley–Jopling classification links immunity to a spectrum of clinical and histopathological expression and remains essential for understanding leprosy as a disease continuum. [2] At one pole, tuberculoid disease shows robust cell-mediated immunity and localized lesions; at the other pole, lepromatous disease reflects poor cell-mediated containment with widespread lesions and higher bacillary load. Intermediate borderline forms are immunologically unstable.

Treatment-oriented classification and operational methods

Because detailed immunologic and histologic classification may not always be feasible, methods for classification for treatment purposes have been described to support standardized therapeutic decisions in public health settings. [3] Such operational approaches guide multidrug therapy but do not replace the deeper clinicopathological understanding provided by spectrum-based classification.

Clinicians should also recognize that classification may shift over time, especially around reactional episodes or immune changes, which can create diagnostic confusion.

HISTOPATHOLOGICAL BASIS AND THE ROLE OF BIOPSY

Histopathology remains a cornerstone for confirming leprosy and defining spectrum. In lepromatous disease, biopsy findings include macrophage-rich infiltrates and characteristic patterns that support diagnosis and clinicopathological correlation. A focused review on histopathology of lepromatous skin biopsy provides a detailed framework for interpreting these patterns. [12]

In clinical practice, additional resources highlight practical diagnostic considerations, including limitations of laboratory access, the importance of correlating clinical findings with pathology, and the need to avoid misdiagnosis in atypical cases. [13] However, biopsy is invasive and may be limited by sampling error (inactive lesion sites), patient acceptability, and availability of trained dermatopathology services. These limitations provide the rationale for adjunctive, non-invasive tools that improve suspicion, support differential diagnosis, and guide biopsy site selection.

LEPROSY REACTIONS:

Clinical Importance and Inflammatory Biology

Leprosy reactions are acute immunological events superimposed on chronic disease and are major contributors to nerve damage and disability. Type 1 (reversal) reaction is particularly linked to borderline disease and is strongly associated with neuritis and disability; epidemiological work emphasizes its importance in disability pathways. [14]

Reactional states are also supported by immunological and cytokine studies; elevated pro-inflammatory mediators have been documented during reactional episodes, reinforcing the immune-driven nature of these events. [15] Clinically, reactions can mimic other inflammatory disorders and may present with abrupt changes in lesions, pain, erythema, edema, and systemic symptoms. Additionally, leprosy can mimic other autoimmune and inflammatory diseases, further complicating diagnosis; reports describing leprosy mimicking lupus erythematosus illustrate this challenge.

Because reactions are time-sensitive and can rapidly worsen neuropathy, any bedside method that helps identify reactional inflammation or lesion activity has practical value.

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DERMATOSCOPY: PRINCIPLES RELEVANT TO HANSEN'S DISEASE

Dermatoscopy is a non-invasive, in vivo technique that enhances visualization of epidermal and superficial dermal structures. Reviews of dermoscopy in general dermatology emphasize its expanding role beyond tumors into inflammatory and infectious dermatoses and discuss future prospects for broader clinical adoption. [16] Dermatoscopy has also been specifically reviewed in the context of cutaneous granulomatous disorders, where recurring themes such as orange-yellow areas and vascular patterns are emphasized. [17]

Polarized vs non-polarized dermatoscopy

Polarized and non-polarized dermoscopy provide complementary information. The explanation for observed differences underscores that polarized dermoscopy enhances deeper dermal visualization and shiny white structures, while non-polarized mode improves surface feature assessment such as scaling. [18] In leprosy, where both surface xerosis/scaling and dermal granulomatous inflammation may be present, combined-mode evaluation improves pattern recognition.

Polarized light vs immersion contact dermoscopy

Practical differences between polarized light dermoscopy and immersion contact dermoscopy influence the visibility of certain structures, especially vessels and shiny white features. Comparative evaluations highlight that technique and modality selection can alter interpretation of vascular patterns. [19] Since pressure can compress vessels and obscure vascular morphology, gentle technique is particularly relevant in leprosy lesions where vascular patterns may have diagnostic value.

Appendageal assessment and hair signs

Hair and follicular changes can be informative in leprosy, particularly when appendageal structures are reduced or altered. While trichoscopy is primarily a hair diagnostic technique, its framework for interpreting hair shaft and follicular signs supports structured assessment of hair-related dermatoscopic findings when evaluating leprosy lesions. [20]

DERMATOSCOPY IN HANSEN'S DISEASE: SPECTRUM-BASED FINDINGS

Evidence describing dermatoscopy in leprosy has grown substantially, including prospective observational studies, clinicopathological correlation studies, and multicentre cross-sectional work. A prospective study directly correlating dermatoscopy with clinical spectrum and histopathology provides key evidence for recurring patterns across subtypes. [21] A separate clinicopathological correlation study further strengthens the association between dermatoscopic features and histology. [22] A multicentre cross-sectional evaluation expands generalizability and provides broader pattern documentation. [23] Additional studies focused on specific spectrum subtypes and clinicopathological correlation complement these findings [FIGURE-1]. [24,25]

Across studies, four domains are consistently emphasized:

1. granuloma-related color/structureless areas (often yellow–orange),
2. focal white areas/white shiny structures (often fibrosis/collagen-related),
3. pigment network disruption and background color changes, and
4. vascular morphology and appendageal/follicular changes.

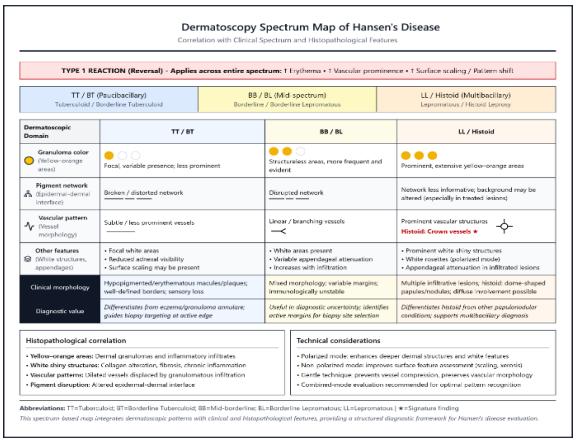

Figure 1. Spectrum-based schematic representation of dermatoscopic patterns in Hansen's disease highlighting changes across

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TT/BT, borderline forms, and LL/histoid leprosy, with an overlay of type 1 reaction-associated inflammatory shifts. [21–23,26,27,29].

Core patterns reported across the spectrum

Yellow–orange structureless areas are repeatedly described and interpreted as dermal granulomas and associated inflammatory infiltrates. [21–23] These areas may be more evident with careful technique and may vary with lesion depth and activity.

Pigment network changes include broken, distorted, or reduced network, often corresponding clinically to hypopigmented or erythematous macules/patches and reflecting altered epidermal–dermal interface. [21,22]

Focal white areas / white shiny structures are reported across multiple subtypes and are interpreted as collagen alteration, fibrosis, or dermal structural change related to chronic inflammation. [21,23]

Vascular patterns—particularly linear and branching vessels—are frequently described and are attributed to dilated vessels displaced by granulomatous infiltration. [21–23]

Appendageal changes (reduced hair follicle density, follicular plugging, altered eccrine openings) are variably reported and may reflect adnexal involvement and trophic changes. [21,25]

9.2 Tuberculoid and borderline tuberculoid spectrum (TT/BT)

In tuberculoid spectrum lesions, dermatoscopy often demonstrates:

- **broken/distorted pigment network**,
- **focal white areas**,
- **surface scaling**, and
- **reduced adnexal visibility** in some lesions. [21,22,25]

A study focused on borderline tuberculoid leprosy highlights the practical role of dermatoscopy in recognizing pigmentary and structural changes that can support diagnosis in clinically confusing plaques. [24] In clinicopathological correlation work, these patterns align with well-formed granulomas and low bacillary load, supporting spectrum-based interpretation. [22]

9.3 Borderline spectrum (BB/BL)

Borderline lesions display mixed features reflecting immunological instability. Dermatoscopy commonly reveals yellow–orange structureless areas and pigment network disruption, with more appreciable vascular patterns than in some tuberculoid lesions. [21–23] Because borderline lesions can resemble other granulomatous or inflammatory dermatoses, dermatoscopy is particularly useful in raising suspicion and guiding biopsy toward active margins. [21,25]

Lepromatous spectrum (LL) and histoid leprosy

Lepromatous disease often shows prominent granuloma-related structureless areas and vascular patterns, along with white shiny structures suggesting dermal fibrosis and chronic infiltrate. [21–23] Appendageal reduction may be more evident in infiltrated lesions, especially when longstanding. [25]

Histoid leprosy is a **distinct** clinical variant characterized by dome-shaped papules/nodules. A clinicodermoscopic case series describes practical dermatoscopic findings that support recognition of histoid lesions. [26] Case-based dermatoscopic descriptions emphasize **crown vessels** and **shiny white structures** in histoid leprosy, patterns that can help distinguish histoid lesions from other nodular dermatoses. [27]

White rosettes and shiny white structures

Rosettes and other white shiny structures are best appreciated under polarized dermatoscopy. Their optical basis and histological correlate have been explored, supporting more confident interpretation when such features are seen in leprosy lesions—particularly in lepromatous/histoid variants where fibrosis and altered collagen are more pronounced. [28]

Dermatoscopy in reactional states

Dermatoscopy can also assist in identifying reactional inflammation. A dedicated report describing dermoscopy of type 1 lepra reaction in skin of color provides evidence of enhanced inflammatory features and pattern shifts that may support early recognition of reactions. [29] Given the disability risk associated with neuritis in type 1

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reactions, any adjunct that improves early suspicion can be clinically meaningful. [14].

CORRELATION OF DERMATOSCOPY WITH HISTOPATHOLOGY

Dermatoscopic changes in leprosy have biologically plausible histopathological correlates:

Yellow–orange structureless areas correspond to dermal granulomas and inflammatory infiltrate. A broader dermatoscopic principle is that orange coloration is a clue for granulomatous diseases, supporting interpretation of these hues as granuloma-related. [30]

Linear/branching vessels reflect dilated dermal vessels displaced upward by infiltrate and altered dermal matrix. Spectrum-correlation studies in leprosy support this interpretation through clinico-dermoscopic-histological comparison. [21,22]

White shiny structures / focal white areas correlate with fibrosis or altered collagen; rosette/shiny white structure studies provide a histological and optical basis for such dermatoscopic findings under polarized light. [28]

Appendageal attenuation and follicular changes may correlate with adnexal involvement and trophic alterations, aligning with the known pathology of leprosy in skin and nerves. [10–12,25]

Overall, correlation studies emphasize that dermatoscopy does not replace biopsy but can strengthen pre-biopsy probability, improve lesion selection for sampling, and enhance clinicopathological correlation [Table-1]. [21,22]

Table 1. Dermatoscopic signs in leprosy and proposed histopathological correlates.

Dermatoscopic feature	Likely histopathological correlate	Interpretation / clinical meaning	Key supporting references
Yellow–orange structureless areas	Dermal granulomas and inflammatory infiltrate	Suggests granulomatous dermatosis; supports leprosy in correct clinical setting	[21–23,30]
Focal white areas / white shiny structures	Fibrosis/altered dermal collagen; remodeled dermis	More prominent in chronic/infiltrated lesions; supports leprosy spectrum and histoid/LL patterns	[21,23,28]
Linear / branching vessels	Dilated dermal vessels displaced upward by granulomas/infiltrate	Marker of active dermal inflammation/infiltration	[21–23]
Pigment network disruption (broken/distorted/reduced)	Altered epidermal–dermal interface; basal pigment disturbance over infiltrate	Supports macular/plaque spectrum lesions; useful in early lesions and skin of color	[21,22,25]
Appendageal attenuation (reduced follicle density, fewer openings)	Adnexal involvement/atrophy or destruction; trophic change	Supports leprosy when combined with granulomatous hue and clinical sensory loss	[10–12,21,25]
Rosettes (polarized)	Optical phenomenon linked	Supportive clue (not specific);	[28]

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	to follicular/adnexal keratin/collagen arrangement	useful in histoid/LL contexts	
"Inflammatory surge" patterns in Type 1 reaction	Acute dermal edema + inflammation in existing lesions	Helps flag reactional activity (especially in skin of color)	[14,29]

DIFFERENTIAL DIAGNOSIS AND OVERLAPS WITH OTHER GRANULOMATOUS DERMATOSES

A limitation of dermatoscopy in leprosy is that granulomatous dermatoses share overlapping dermatoscopic themes, particularly orange-yellow areas and vascular patterns. Granuloma annulare, for example, has been studied with dermoscopy and histological correlation, demonstrating features that can overlap with leprosy. [31] Dermoscopy of necrobiosis lipoidica and granuloma annulare also illustrates shared granulomatous hues and vascular configurations. [32]

Cutaneous sarcoidosis and necrobiotic granulomas may show overlapping dermatoscopic patterns; work suggests dermoscopy can assist in differentiating sarcoidosis from necrobiotic granulomas, even after treatment. [33] Additional descriptions of dermoscopy in cutaneous sarcoidosis further support the diagnostic value and also underscore potential overlap with other granulomatous conditions. [34]

Because "orange" is a general granulomatous clue rather than a leprosy-specific sign, leprosy suspicion relies on combinations of findings (granulomatous hues plus pigment network disruption, appendageal reduction, follicular plugging patterns, and reaction-associated surface changes) interpreted in clinical context (sensory loss, nerve thickening, distribution patterns). [21–23,25,30]

Leprosy can also clinically mimic autoimmune dermatoses, including lupus-like presentations; such cases highlight the importance of considering leprosy in differential diagnosis and the potential role of dermatoscopy in supporting earlier suspicion [Table-2]. [35].

Table 2. Dermatoscopic differentials: leprosy versus common granulomatous mimickers

Condition	Shared dermatoscopic overlap with leprosy	Dermatoscopic clues favoring the condition	Take-home differentiation point	References
Granuloma annulare	Orange-yellow areas; vessels may be seen	Patterned annular distribution; disease-specific GA features on dermoscopy	Orange hue alone not diagnostic; interpret with morphology and sensory loss	[31,32]
Necrobiosis lipoidica	Orange-yellow areas; prominent vessels	Serpiginous/"network" vessels with atrophic plaque phenotype	Strong vascular pattern with classic plaque setting suggests NL	[32]
Cutaneous sarcoidosis	Orange-yellow areas; vascular prominence	Dermoscopy helpful but overlap substantial; context critical	Requires clinicopathological correlation; leprosy clues include appendag	[33,34]

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			eal loss + sensory change	
Granulomatous disorders (general)	"Orange color" as a common clue	Orange is a general granulomatous marker rather than disease-specific	Combination of signs + clinical exam decides	[17,30]
Leprosy (Hansen's disease)	Orange-yellow granulomatous hue possible	Pigment network disruption + appendageal attenuation + reactional changes; supported by spectrum patterns	Dermoscopy supports suspicion; confirmation via biopsy/SSS in uncertain cases	[21–23,25,29]

CLINICAL APPLICATIONS OF DERMATOSCOPY IN LEPROSY

Dermatoscopy can add value at several points in clinical workflow:

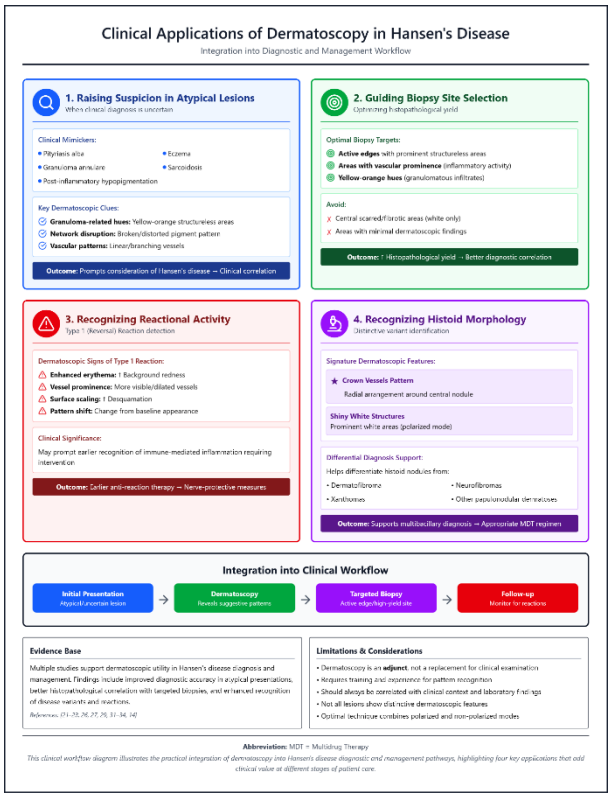
Raising suspicion in atypical lesions: When lesions resemble pityriasis alba, eczema, granuloma annulare, sarcoidosis, or post-inflammatory hypopigmentation, dermatoscopy may reveal granuloma-related hues, network disruption, and vascular patterns that prompt consideration of leprosy. [21–23,31–34]

Guiding biopsy site selection: Choosing active edges with prominent structureless areas and vascular clues may increase histopathological yield and strengthen correlation. [21,22]

Supporting recognition of reactional activity: Dermatoscopy may highlight enhanced erythema, scaling, and vessel prominence in type 1 reactions, potentially prompting earlier anti-reaction therapy and nerve-protective measures. [29,14]

Recognizing histoid morphology: Crown vessels and shiny white structures may support recognition of histoid nodules and differentiation from other papulonodular dermatoses. [26,27].

Figure 2. Clinical applications of dermatoscopy in



Hansen's disease and integration into the diagnostic–management workflow. The figure summarizes four practical roles of dermatoscopy: (1) raising suspicion in atypical lesions by identifying granuloma-related yellow–orange structureless areas, pigment network disruption, and characteristic vascular patterns; (2) guiding biopsy site selection by targeting active edges with prominent structureless areas and vascular prominence to optimize histopathological yield; (3) recognizing reactional activity (type 1/reversal reaction) through enhanced erythema, increased vessel visibility, surface scaling, and pattern shift; and (4) supporting identification of histoid leprosy via crown vessels and shiny white structures on polarized mode. A simplified pathway illustrates incorporation of dermatoscopy from initial presentation to targeted biopsy and follow-up for reactions. References: [14, 21–23,26,27,29,31–34].

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RESEARCH GAPS AND FUTURE DIRECTIONS

Despite encouraging evidence, several gaps remain:

Standardized terminology and reporting: Studies use varied descriptors and methods; standardized definitions for color tones, structureless areas, vascular morphologies, and appendageal metrics would improve reproducibility. [17,21–23]

Spectrum-stratified validation: Larger multicentre studies stratified by Ridley–Jopling subtype, bacterial index, lesion duration, and skin phototype are needed to evaluate diagnostic performance. [21–23]

Reaction-focused characterization: Type 1 and type 2 reactions need systematic dermatoscopic characterization linked to clinical neuritis outcomes and histopathology. [14,15,29]

Diagnostic accuracy studies: Most evidence is descriptive; future work should assess sensitivity/specificity of defined dermatoscopic criteria for leprosy versus granulomatous mimickers. [30–34]

Digital dermatoscopy and pattern libraries: Standardized image repositories may enable more consistent teaching and could support AI-assisted triage, particularly in resource-limited settings.

CONCLUSION

Dermatoscopy is a practical, non-invasive adjunct that can support the recognition and classification of Hansen's disease by demonstrating granuloma-associated color changes, pigment network disruption, vascular remodeling, and appendageal alterations. Prospective, clinicopathological correlation, and multicentre studies consistently describe patterns that correlate with clinical spectrum and histopathology. While dermatoscopy does not replace SSS or biopsy, it can refine differential diagnosis, guide biopsy site selection, and help identify reactional activity, potentially reducing diagnostic delay and preventing disability. Future research should prioritize standardization and accuracy-based validation across diverse populations and clinical settings.

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REFERENCES

1. Scollard DM, Adams LB, Gillis TP, Krahenbuhl JL, Truman RW, Williams D. The continuing challenges of leprosy. Clinical Microbiology Reviews. 2006;19(2):338-81.
2. Ridley DS, Jopling WH. Classification of leprosy according to immunity. A five-group system.
3. Pardillo FE, Fajardo TT, Abalos RM, Scollard D, Gelber RH. Methods for the classification of leprosy for treatment purposes. Clinical Infectious Diseases. 2007;44(8):1096-9.
4. Global leprosy situation, 2012. Wkly Epidemiol Rec. 2012;87(34):317-28.
5. Global leprosy situation, 2010. Wkly Epidemiol Rec. 2010;85(35):337-48.
6. Truman RW, Singh P, Sharma R, Busso P, Rougemont J, Paniz-Mondolfi A, et al. Probable zoonotic leprosy in the southern United States. New England Journal of Medicine. 2011;364(17):1626-33.
7. Truman R. Leprosy in wild armadillos. Leprosy Review. 2005;76(3):198-208.
8. Cole ST, Eiglmeier K, Parkhill J, James KD, Thomson NR, Wheeler PR, et al. Massive gene decay in the leprosy bacillus. Nature. 2001;409(6823):1007-11.
9. 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. PNAS. 2015;112(14):4459-64.
10. Bhat RM, Prakash C. Leprosy: an overview of pathophysiology. Interdisciplinary Perspectives on Infectious Diseases. 2012;2012:181089.
11. Adams LB, Krahenbuhl JL. Human leprosy. In: Granulomatous Infections and Inflammations: Cellular and Molecular Mechanisms. 2003:207-44.

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JCHR (2026) 16(1), 1252-1262 | ISSN:2251-6727

12. Massone C, Belachew WA, Schettini A. Histopathology of the lepromatous skin biopsy. Clinics in Dermatology. 2015;33(1):38-45.

13. Bhandari J, Awais M, Gupta V. Leprosy (Hansen Disease). StatPearls [Internet]. 2020.

14. Lienhardt C, Fine PE. Type 1 reaction, neuritis and disability in leprosy. Leprosy Review. 1994;65(1):9-33.

15. Sarno EN, Grau GE, Vieira LM, Nery JA. Serum levels of tumour necrosis factor-alpha and interleukin-1 beta during leprosy reactional states. Clinical and Experimental Immunology. 1991;84(1):103.

16. 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;9(4):253.

17. Chauhan P, Adya KA. Dermatoscopy of cutaneous granulomatous disorders. Indian Dermatology Online Journal. 2021;12(1):34-44.

18. Pan Y, Gareau DS, Scope A, Rajadhyaksha M, Mullani NA, Marghoob AA. Polarized and nonpolarized dermoscopy: the explanation for the observed differences. Archives of Dermatology. 2008;144(6):828-9.

19. Benvenuto-Andrade C, Dusza SW, Agero AL, Scope A, Rajadhyaksha M, Halpern AC, Marghoob AA. Differences between polarized light dermoscopy and immersion contact dermoscopy for the evaluation of skin lesions. Archives of Dermatology. 2007;143(3):329-38.

20. Rudnicka L, Olszewska M, Rakowska A, Kowalska-Oledzka E, Slowinska M. Trichoscopy: A new method for diagnosing hair loss. J Drugs Dermatol. 2008;7:651–4.

21. 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;33(10):1947-51.

22. 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;11(2):e2021032.

23. Ankad BS, Sharma A, Vinay K, Rathod S, Mehta H, Bhat YJ, Ashwini PK, Jha AK, Narang T. Dermatoscopic evaluation of leprosy: A multi-centre cross-sectional study. Indian Journal of Dermatology, Venereology and Leprology. 2024;90(4):486-93.

24. Ankad BS, Sakhare PS. Dermoscopy of borderline tuberculoid leprosy. International Journal of Dermatology. 2018;57(1):74-6.

25. Bhatia AS, Badad A, Hogade AS, Spoorthy M. Dermatoscopy and clinicopathological correlation in different spectrum of leprosy. Clinical Dermatology Review. 2021;5(1):65-70.

26. Acharya P, Mathur MC. Clinicodermoscopic study of histoid leprosy: a case series. International Journal of Dermatology. 2020;59(3):365-8.

27. Abadías-Granado I, Navarro-Bielsa A, Gómez-Mateo MC, Bermúdez-Cameo R, Gilaberte Y. Crown vessels and shiny white structures in dermoscopy of histoid leprosy. JAAD Case Reports. 2020;6(11):1147-9.

28. Haspeslagh M, Noë M, De Wispelaere I, Degryse N, Vossaert K, Lanssens S, Facchetti F, Van Den Bossche K, Brochez L. Rosettes and other white shiny structures in polarized dermoscopy: histological correlate and optical explanation. Journal of the European Academy of Dermatology and Venereology. 2016;30(2):311-3.

29. Jha AK, Zeeshan MD, Tiwary P, Singh A, Roy PK, Chaudhary RK. Dermoscopy of type 1 lepra reaction in skin of color. Dermatology Practical & Conceptual. 2020;10(3):e2020083.

30. Bombonato C, Argenziano G, Lallas A, Moscarella E, Ragazzi M, Longo C. Orange color: a dermoscopic clue for the diagnosis of granulomatous skin diseases. Journal of the American Academy of Dermatology. 2015;72(1):S60-3.

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31. Errichetti E, Lallas A, Apalla Z, Di Stefani A, Stinco G. Dermoscopy of granuloma annulare: a clinical and histological correlation study. Dermatology. 2017;233(1):74-9.

32. Pellicano R, Caldarola G, Filabozzi P, Zalaudek I. Dermoscopy of necrobiosis lipoidica and granuloma annulare. Dermatology. 2013;226(4):319-23.

33. Ramadan S, Hossam D, Saleh MA. Dermoscopy could be useful in differentiating sarcoidosis from necrobiotic granulomas even after treatment with systemic steroids. Dermatology Practical & Conceptual. 2016;6(3):17.

34. Loo Lim AY, Tang PY, Oh CC. Dermoscopy in cutaneous sarcoidosis. Dermatology Online Journal. 2021;27(9).

35. Damodar SS, Smitha P, Nirmal B, Sudhir NU, Ballambat PS. Hansen's disease associated with erythromelalgia mimicking lupus erythematosus. Indian Dermatology Online Journal. 2014;5(1):59-62.