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Clinicopathological features of hypopigmentary skin lesions and role of Melan: A an immunohistochemistry marker: A cross-sectional study

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Abstract

Background: Hypopigmentary skin lesions comprise a diverse group of dermatological disorders resulting from alterations in melanocyte number, melanin synthesis, or melanin transfer. Because many of these conditions exhibit overlapping clinical features, accurate diagnosis often requires histopathological examination and ancillary immunohistochemical techniques. Melan-A immunohistochemistry has emerged as a valuable tool for evaluating melanocyte density and differentiating melanocytopenic from melanopenic disorders.

Aim: To study the clinicopathological spectrum of hypopigmentary skin lesions and evaluate the role of Melan-A immunohistochemistry in their subclassification. Melan-A immunohistochemistry in their subclassification.

Methodology: This cross-sectional study was conducted on 60 clinically diagnosed cases of hypopigmentary skin lesions. Detailed clinical evaluation was followed by skin biopsy and histopathological examination using routine hematoxylin and eosin staining. Clinicopathological correlation was performed in all cases. Melan-A immunohistochemistry was applied to assess melanocyte density and classify lesions into melanocytopenic and melanopenic categories. Data were analyzed using descriptive statistical methods.

Results: Among the 60 patients studied, females constituted 60% and males 40% of the study population. The majority of patients belonged to the 21–30-year age group (43%). Extremities were the most commonly involved site (55%), followed by the face (17%). Hansen's disease was the most frequent clinical diagnosis, accounting for 26 cases (44%), and remained the most common histopathological diagnosis with 25 cases (42%). Pityriasis lichenoides chronica constituted 8 cases (14%), while post-inflammatory hypopigmentation accounted for 6 cases (10%) on histopathological examination. Histopathological analysis demonstrated characteristic findings including granuloma formation in Hansen's disease, basal cell vacuolar degeneration in pityriasis lichenoides chronica and lichen sclerosus et atrophicus, pigment incontinence in pityriasis lichenoides chronica and post-inflammatory hypopigmentation, and reduced melanin pigment in the majority of lesions. Clinicopathological correlation showed excellent agreement, with only three discordant cases identified. Melan-A immunohistochemistry demonstrated melanocytopenia in 6 cases (10%), comprising idiopathic guttate hypomelanosis, vitiligo, and one case of pityriasis lichenoides chronica, whereas 54 cases (90%) were classified as melanopenic lesions with preserved melanocyte populations.

Conclusion: Histopathological examination remains the cornerstone for definitive diagnosis of hypopigmentary skin lesions. Melan-A immunohistochemistry serves as a valuable adjunct in differentiating melanocytopenic from melanopenic disorders, improving diagnostic accuracy and strengthening clinicopathological correlation.

Keywords: Hypopigmentary skin lesions; Histopathology; Melan-A; Clinicopathological Correlation; Immunohistochemistry

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1. Introduction

Hypopigmentary skin lesions constitute a heterogeneous group of dermatological disorders characterized by partial or complete reduction in skin pigmentation. These lesions may result from a decrease in melanin synthesis, defective melanosome transfer, reduced melanocyte activity, or actual loss of melanocytes from the epidermis. Clinically, hypopigmented lesions encompass a wide spectrum of inflammatory, infectious, autoimmune, genetic, and degenerative disorders, many of which demonstrate overlapping morphological features and therefore pose significant diagnostic challenges.[1]

Accurate diagnosis of hypopigmentary disorders is essential because therapeutic strategies, prognosis, and long-term outcomes vary considerably depending on the underlying pathology. Although clinical examination remains the cornerstone of diagnosis, considerable overlap exists among various hypopigmented dermatoses including Hansen's disease, vitiligo, pityriasis lichenoides chronica, lichen sclerosus et atrophicus, post-inflammatory hypopigmentation, pityriasis versicolor, idiopathic guttate hypomelanosis, and connective tissue disorders.[2] Consequently, reliance solely on clinical findings may lead to diagnostic uncertainty and inappropriate management.

Histopathological examination remains the gold standard for definitive diagnosis of many hypopigmentary skin lesions. Skin biopsy enables evaluation of epidermal architecture, melanocyte distribution, inflammatory infiltrates, granuloma formation, pigment incontinence, collagen alterations, and dermoepidermal interface changes, thereby facilitating differentiation among clinically similar conditions.[3] Histopathology is particularly useful in disorders such as Hansen's disease, lichen sclerosus et atrophicus, discoid lupus erythematosus, pityriasis lichenoides chronica, and morphea, where characteristic microscopic features often provide the definitive diagnosis.[4]

Among hypopigmentary disorders, Hansen's disease continues to represent an important cause of acquired hypopigmentation in developing countries. Histopathological evaluation plays a crucial role not only in confirming the diagnosis but also in subclassifying the disease and assessing disease activity.[5] Similarly, inflammatory disorders such as pityriasis lichenoides chronica and post-inflammatory hypopigmentation frequently demonstrate overlapping clinical appearances, necessitating tissue examination for accurate diagnosis.[6]

The pathogenesis of hypopigmentation may be broadly categorized into melanocytopenic and melanopenic mechanisms. Melanocytopenic lesions are characterized by a reduction or absence of melanocytes, whereas melanopenic lesions retain melanocytes but exhibit decreased melanin production or impaired melanosome transfer. Distinguishing between these mechanisms is often difficult on routine hematoxylin and eosin staining alone, particularly in subtle or early lesions.[7]

Immunohistochemistry has emerged as a valuable adjunct in dermatopathology for identifying specific cellular populations and improving diagnostic precision. Among available melanocytic markers, Melan-A (MART-1) is a highly sensitive and specific marker for melanocytes. It highlights melanocytic cytoplasm and permits accurate assessment of melanocyte density within the basal epidermal layer.[8] Melan-A immunostaining has therefore become an important tool for evaluating pigmentary disorders and differentiating true melanocyte loss from disorders characterized primarily by altered melanin production. [9]

Previous studies have demonstrated the usefulness of Melan-A in evaluating vitiligo, idiopathic guttate hypomelanosis, post-inflammatory hypopigmentation, and other pigmentary disorders. In melanocytopenic lesions such as vitiligo, Melan-A demonstrates marked reduction or complete absence of melanocytes, whereas melanopenic disorders generally show preservation of melanocyte numbers despite clinically evident hypopigmentation.[10] Such differentiation has significant diagnostic and pathogenetic implications and may aid in classification of hypopigmentary dermatoses.

Despite the increasing utility of immunohistochemistry, relatively few studies have comprehensively evaluated the clinicopathological spectrum of hypopigmentary skin lesions while simultaneously assessing the role of Melan-A immunostaining in lesion subclassification. The present study was therefore undertaken to analyze the clinicopathological features of hypopigmentary skin lesions and to evaluate the role of Melan-A immunohistochemistry in differentiating melanopenic from Melano cytopenic disorders. Such an approach may contribute to improved diagnostic accuracy, better understanding of disease pathogenesis, and more effective patient management.

Aim

To study clinicopathological features of hypopigmentary skin lesions and evaluate the role of Melan-A immunohistochemistry marker.

2. Methodology

The present cross-sectional observational study was conducted in the Department of Pathology in collaboration with the Department of Dermatology over a defined study period after obtaining approval from the Institutional Ethics Committee. The study included patients clinically presenting with hypopigmentary skin lesions who underwent skin biopsy for histopathological evaluation. A total of 60 biopsy specimens were included in the study after application of inclusion and exclusion criteria. Detailed demographic information including age, sex, duration of lesions, site of involvement, clinical presentation, associated symptoms, and provisional clinical diagnosis was recorded in all cases. Patients presenting with hypopigmented macules, patches, plaques, or diffuse hypopigmentation were included in the study, whereas inadequate biopsy specimens, poorly preserved tissue samples, previously treated lesions with severe secondary changes, and patients unwilling to participate were excluded.

Punch biopsy or excisional biopsy specimens were obtained under aseptic precautions and fixed in 10% buffered formalin. Tissue processing was performed according to standard histopathological protocols followed by paraffin embedding. Sections of 4–5 µm thickness were prepared and stained with hematoxylin and eosin stain for routine microscopic examination. Histopathological evaluation was performed with special emphasis on epidermal changes, basal pigmentation, melanocyte distribution, inflammatory infiltrate, dermal alterations, pigment incontinence, fungal elements, adnexal structures, and associated pathological findings. Morphological diagnosis was established based on clinicopathological correlation.

Immunohistochemical staining for Melan-A was performed in all study cases to assess melanocyte distribution and density. Sections were mounted on poly-L-lysine-coated slides followed by deparaffinization, rehydration, antigen retrieval, blocking of endogenous peroxidase activity, and incubation with primary Melan-A antibody. Secondary antibody application and chromogenic detection were subsequently performed according to standardized immunohistochemical protocols. Appropriate positive and negative controls were included in all staining runs. Melan-A expression was evaluated based on melanocyte distribution along the basal epidermal layer, staining intensity, continuity of melanocytic lining, and percentage of stained melanocytes.

Clinicopathological correlation was carried out to compare clinical diagnosis, routine histopathological findings, and immunohistochemical features. Lesions were categorized into melanocytopenic and melanopenic disorders based on histomorphological and immunohistochemical findings. Statistical analysis was performed using SPSS software version 26.0. Descriptive statistics including frequency, percentage, were calculated. Chi-square test was applied for comparison of categorical variables and p-value <0.05 was considered statistically significant. Confidentiality of patient information was maintained throughout the study.

Detailed clinicopathological evaluation, histopathological examination, and Melan-A immunohistochemical analysis were performed in all cases.

3. Results

A total of 60 patients with clinically diagnosed hypo pigmentary skin lesions were included in the study. Detailed clinicopathological evaluation, histopathological examination, and Melan-A immunohistochemical analysis were performed in all cases.

Table 1 Age-wise Distribution of Cases (n = 60)

Variable	Category	Number of Cases	Percentage (%)
Age Group (years)	0–10	4	7
	11–20	18	30
	21–30	26	43
	31–40	8	13
	41–50	4	7

The majority of patients belonged to the 21–30-year age group (43%), followed by the 11–20-year age group (30%).

Table 2 Distribution of lesions based on histopathological examination

S.No	Histopathological Diagnosis	(No. Of Cases)	Percentage(%)
1	Pityriasis lichenoides chronica	8	14
2	Hansen's disease	25	42
3	Vitiligo	2	4
4	Lichen sclerosus et atrophicus	5	7
5	Idiopathic guttate hypomelanosis	3	5
6	Lichen striatus	3	5
7	Pityriasis versicolor	2	3
8	Amyloidosis cutis dyschromica	3	5
9	Discoid lupus erythematosus	2	3
10	Post inflammatory hypopigmentation	6	10
11	Morphea	1	2
	Total	60	100

Histopathological examination of the 60 biopsied cases demonstrated a broad spectrum of hypopigmentary dermatoses. Hansen's disease was the most common histopathological diagnosis, confirmed in 25 cases (42%), followed by pityriasis lichenoides chronica in 8 cases (14%). Post-inflammatory hypopigmentation accounted for 6 cases (10%), while lichen Sclerosus et atrophicus was identified in 5 cases (7%). Idiopathic guttate hypomelanosis, lichen striatus, and amyloidosis cutis dyschromica were each observed in 3 cases (5%). Vitiligo and discoid lupus erythematosus were diagnosed in 2 cases (3–4%) each, whereas pityriasis versicolor was confirmed in 2 cases (3%). Morphea was the least common lesion, accounting for 1 case (2%). Overall, the histopathological findings closely correlated with the clinical diagnoses and highlighted Hansen's disease as the predominant cause of hypopigmentary skin lesions in the study population, followed by pityriasis lichenoides chronica and post-inflammatory hypopigmentation. Histopathological evaluation proved essential in establishing definitive diagnosis and differentiating clinically overlapping hypo pigmentary disorders.

Table 3 Major Histopathological Findings and Clinicopathological Correlation

Histopathological Feature	Predominant Lesions
Granuloma formation	Hansen's disease (14 cases)
Thin epidermis	Hansen's disease (12 cases), LSEA (5 cases)
Basal cell vacuolar degeneration	PLC (8 cases), LSEA (5 cases), DLE (2 cases)
Pigment incontinence	PLC (6 cases), PIH (5 cases)
Reduced melanin pigment	Hansen's disease (25 cases), PLC (8 cases), PIH (5 cases)
Absent melanocytes and melanin	Vitiligo (2 cases)
Reduced melanocytes	IGH (3 cases), PLC (1 case)

Clinicopathological correlation demonstrated excellent concordance between clinical and histopathological diagnoses. Among 26 clinically diagnosed cases of Hansen's disease, 25 were confirmed histopathologic ally, while one case was reclassified as post-inflammatory hypopigmentation. One clinically diagnosed case of pityriasis versicolor was identified as lichen sclerosus et atrophicus on histopathology, and one clinically suspected case of lichen sclerosus et atrophicus was diagnosed as morphea. Overall, only three discordant cases were identified, indicating a high degree of clinicopathological agreement.

Table 4 Subclassification of Hypo pigmentary Skin Lesions Based on Melan-A Immunohistochemistry (n = 60)

Category	Number of Cases	Percentage (%)
Melanocytopenic (0–4 melanocytes/100 basal keratinocytes)	6	10
Melanopenic (>5 melanocytes/100 basal keratinocytes)	54	90
Total	60	100

Melan-A immunohistochemistry demonstrated melanocytopenia in 6 cases (10%), comprising three cases of idiopathic guttate hypomelanosis, two cases of vitiligo, and one case of pityriasis lichenoides chronica. The remaining 54 cases (90%) retained adequate melanocyte populations and were categorized as melanopenic lesions. These included Hansen's disease, lichen sclerosus et atrophicus, post-inflammatory hypopigmentation, pityriasis versicolor, amyloidosis cutis dyschromica, discoid lupus erythematosus, morphea, and the majority of pityriasis lichenoides chronica cases.

The findings indicate that most hypopigmentary dermatoses in the study population resulted from altered melanin production, pigment transfer abnormalities, or inflammatory pigmentary changes rather than actual melanocyte depletion.

4. Discussion

The present study evaluated 60 cases of hypopigmentary skin lesions with the aim of assessing their clinicopathological spectrum and determining the role of Melan-A immunohistochemistry in differentiating melanopenic from melanocytopenic disorders. The analysis incorporated histopathological diagnosis, characteristic microscopic findings, clinicopathological correlation, and Melan-A-based subclassification of lesions.

In the present study, histopathological examination revealed a broad spectrum of hypo pigmentary dermatoses. Hansen's disease was the most common diagnosis, accounting for 25 cases (42%), followed by pityriasis lichenoides chronica in 8 cases (14%). Post-inflammatory hypopigmentation constituted 6 cases (10%), while lichen sclerosis et atrophicus was identified in 5 cases (7%). Less frequent lesions included idiopathic guttate hypo melanosis, lichen striatus, amyloidosis cutis dyschromic, vitiligo, discoid lupus erythematosus, pityriasis versicolor, and morphea.

The predominance of Hansen's disease observed in the present study is comparable with observations reported by Sehgal et al., who identified Hansen's disease as a common cause of hypopigmented lesions in dermatopathology practice. Similar findings were reported by Kar et al., who emphasized the diagnostic importance of skin biopsy in differentiating Hansen's disease from other clinically similar hypo pigmentary disorders. Furthermore, Kumar et al. demonstrated that histopathological evaluation remains indispensable for confirming the diagnosis and subclassification of Hansen's disease. Bhatia et al. also highlighted the wide clinicopathological spectrum of hypopigmented dermatoses and the importance of tissue diagnosis in establishing definitive diagnosis.

The relatively high frequency of pityriasis lichenoides chronica and post-inflammatory hypopigmentation in the present study is consistent with reports by Weedon et al., who described these entities as important differential diagnoses in patients presenting with acquired hypopigmented lesions. Similar observations were reported by Lever and Elder, who emphasized that overlapping clinical morphology often necessitates histopathological confirmation.

The present study demonstrated distinct histopathological patterns across various hypopigmentary disorders. Granuloma formation and lymphohistiocytic infiltrates were characteristic findings in Hansen's disease. Basal cell vacuolar degeneration was frequently observed in pityriasis lichenoides chronica, lichen striatus, lichen sclerosus et atrophicus, and discoid lupus erythematosus. Thickened collagen bundles were identified in lichen sclerosus et atrophicus, discoid lupus erythematosus, and morphea. Reduced melanin pigment was observed in the majority of lesions, whereas actual melanocyte reduction was restricted to vitiligo, idiopathic guttate hypomelanosis, and one case of pityriasis lichenoides chronica.

These findings correlate well with the observations of Lever and Elder, who described granulomatous inflammation as a hallmark feature of Hansen's disease and emphasized the role of histopathology in distinguishing it from inflammatory hypo pigmentary conditions. Weedon similarly reported basal cell vacuolar degeneration as a characteristic finding in interface dermatoses including pityriasis lichenoides chronica and discoid lupus erythematosus.

The presence of pigment incontinence in pityriasis lichenoides chronica and post-inflammatory hypopigmentation observed in the present study corresponds with descriptions by Calonje et al., who noted that pigmentary alteration frequently accompanies interface dermatitis. Likewise, Elder et al. highlighted that reduced melanin content may occur despite preservation of melanocyte numbers in several inflammatory dermatoses, thereby explaining the occurrence of hypopigmentation without actual melanocyte loss.

Clinicopathological correlation revealed a high degree of agreement between clinical and histopathological diagnoses. Among the 26 clinically suspected cases of Hansen's disease, 25 were confirmed histopathologically, while one case was reclassified as post-inflammatory hypopigmentation. One clinically diagnosed case of pityriasis versicolor was histopathologically identified as lichen sclerosus et atrophicus. Similarly, one clinically suspected case of lichen sclerosus et atrophicus was ultimately diagnosed as morphea. All remaining entities demonstrated complete concordance between clinical and histopathological findings.

The strong clinicopathological agreement observed in the present study is comparable with findings reported by Sehgal et al., who demonstrated that histopathological examination substantially improves diagnostic accuracy in hypopigmented dermatoses. Similar observations were reported by Kar et al., who emphasized that histopathology remains the gold standard for confirmation of clinically ambiguous lesions.

Bhatia et al. also documented that several hypopigmentary disorders demonstrate overlapping clinical features, making biopsy evaluation essential for definitive diagnosis. Furthermore, Lever and Elder noted that clinicopathological correlation provides the highest diagnostic reliability when evaluating inflammatory and pigmentary skin disorders. The minimal discordance observed in the present study therefore highlights the effectiveness of combined clinical and histopathological assessment.

A major objective of the present study was to evaluate the role of Melan-A immunohistochemistry in subclassifying hypopigmentary skin lesions. Melan-A staining demonstrated that only 6 cases (10%) showed melanocytopenia, characterized by true reduction or absence of melanocytes. These lesions included idiopathic guttate hypomelanosis, vitiligo, and one case of pityriasis lichenoides chronica. The remaining 54 cases (90%) retained adequate melanocyte populations and were classified as melanopenic lesions.

These findings indicate that most hypopigmentary lesions result from reduced melanin production, altered melanosome transfer, or inflammatory pigmentary alteration rather than actual melanocyte depletion. Similar observations were reported by Orchard et al., who demonstrated the utility of Melan-A immunostaining in accurately identifying melanocyte populations in depigmented and hypopigmented lesions.

The melanocytopenic pattern observed in vitiligo is consistent with findings reported by Le Poole et al., who demonstrated near-complete loss of epidermal melanocytes in established vitiligo lesions. Likewise, studies by Tobin et al. confirmed that melanocyte depletion represents the primary pathological mechanism in vitiligo and certain cases of idiopathic guttate hypomelanosis.

In contrast, preservation of melanocyte numbers in Hansen's disease, post-inflammatory hypopigmentation, pityriasis versicolor, and lichen sclerosus et atrophicus supports the concept that these conditions are predominantly melanopenic. Similar findings were reported by Weedon and Elder, who described decreased melanin synthesis and altered pigment transfer as the major mechanisms responsible for hypopigmentation in inflammatory dermatoses.

The findings of the present study demonstrate that histopathological examination remains indispensable for accurate diagnosis of hypopigmentary skin lesions. Although clinical evaluation provides an initial diagnostic impression, overlapping morphological features frequently necessitate tissue diagnosis. Furthermore, Melan-A immunohistochemistry serves as a valuable adjunct by differentiating melanocytopenic disorders from melanopenic lesions, thereby improving diagnostic precision and facilitating appropriate management.

Overall, the present study highlights the complementary role of clinicopathological correlation and Melan-A immunohistochemistry in the evaluation of hypopigmentary dermatoses. Histopathological examination established definitive diagnosis in the majority of cases, while Melan-A staining provided objective assessment of melanocyte status. Combined utilization of conventional histopathology and immunohistochemistry therefore enhances diagnostic accuracy and contributes significantly to the understanding and classification of hypopigmentary skin disorders.

5. Conclusion

Melan-A immunohistochemistry is a valuable adjunctive diagnostic tool in evaluation of hypopigmentary skin lesions and significantly improves clinicopathological correlation and diagnostic accuracy. Combined histopathological and immunohistochemical assessment facilitates accurate differentiation between melanocytopenic and melanopenic disorders and enhances overall diagnostic reliability.

Limitations of the study

The present study was conducted at a single tertiary care center with a relatively limited sample size, which may restrict generalization of findings to larger populations. Long-term clinical follow-up and assessment of therapeutic response were not included in the study. Additional multicentric studies with larger sample sizes and inclusion of other melanocytic markers may provide further validation of the diagnostic utility of Melan-A immunohistochemistry.

Recommendations

Melan-A immunohistochemistry should be considered in diagnostically challenging hypopigmentary skin lesions showing overlapping clinical and histopathological features. Routine incorporation of melanocytic markers may improve diagnostic precision and facilitate appropriate clinical management. Further comparative studies evaluating additional immunohistochemical markers may help establish standardized diagnostic protocols for hypopigmentary disorders.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of ethical approval

Institutional ethical committee approval was obtained prior to commencement of the study.

Statement of informed consent

Written informed consent was obtained from all participants and confidentiality of patient information was maintained throughout the study period.

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