STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE

BY

REGISTRATION NUMBER BT0122001

DISSERTATION

Submitted to

KAHER, Belagavi, Karnataka.

In partial fulfillment of the requirements for the degree of

M.D.

IN

DEPARTMENT OF DERMATOLOGY, VENEREOLOGY AND LEPROSY

JAWAHARLAL NEHRU MEDICAL COLLEGE, KAHER, BELAGAVI-590010

KARNATAKA

MARCH/APRIL 2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Endorsement by the HOD/ Principal/ Head of the Institution

This is to certify that the dissertation entitled "**STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE**" is bonafide research work done by **BT0122001** in the department of Dermatology, Venereology, and Leprosy, Jawaharlal Nehru Medical College, Nehru Nagar, Belagavi-590010.

DR. BHAVANA R DOSHI MD, DVL, FIDD
Professor and Head
Department of Dermatology
Venereology and Leprosy
J. N. Medical College,
Nehru Nagar, Belagavi- 590010

Date: 26/3/2025
Place: BELAGAVI

DR. N. S. MAHANTSHETTI MD
Principal
J. N. Medical College,
Nehru Nagar, Belagavi- 590010

PRINCIPAL
Jawaharlal Nehru Medical College
BELAGAVI

Date:
Place: BELAGAVI

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

Copyright

Declaration by the candidate

I hereby declare that KAHER, Belagavi, Karnataka shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic/ research purpose.

Date : 26/3/25

Place: BELAGAVI

REG NO: BT0122001

UNDERTAKING

I, **REG NO: BT0122001**, hereby declare that the information and the data mentioned in my dissertation entitled "**STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE**" belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts, or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 26/3/25
Place: BELAGAVI

REG NO: BT0122001

KLE JAWAHARLAL NEHRU MEDICAL COLLEGE
(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)
(Recognized by National Medical Commission, New Delhi)
Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MoE (GoI)
Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA
0831 - 2471350 0831 - 2470759 www.jnmc.edu rincipal@jnmc.edu

Ref No: MDC/PG/ Date: 18-03-2025

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: **"STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE"** has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **05%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER * J.N. MEDICAL COLLEGE * BELAGAVI

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

To,
Reg. No. BT0122001
Postgraduate Student,
2022-23 Batch,
Department of Skin & V.D.
J. N. Medical College, Belagavi.

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 109 **Date: 30/03/2023**

To,
BT0122001
PG Student in Dermatology, Venerology and Leprosy
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS WITH AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

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

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagav

ABSTRACT

BACKGROUND

The emergence of small and large pigmented macules or patches serves as a clinical marker for a range of conditions collectively known as acquired dermal macular hyperpigmentation (ADMH). ADMH aims to encompass conditions that have been acknowledged in prior literature, such as lichen planus pigmentosus, ashy dermatosis, and Riehl's melanosis/pigmented cosmetic dermatitis.

AIMS AND OBJECTIVES

To assess ADMH's dermatoscopic features and determine how they relate to histopathological results.

MATERIAL AND METHODS

A Hospital based cross-sectional study was carried out from July 2023 to July 2024 on 56 patients who were clinically diagnosed with ADMH of face attending Dermatology, Venerology and Leprosy OPD at Dermatology, Venerology and Leprosy, KLE Institute and were willing to participate. Dermoscopy was conducted on the bilateral preauricular and frontal areas, covering four specific sites for facial involvement. A hand-held DermLite DL4 dermo scope was utilized for evaluation. The initial histopathological examination was conducted to confirm the diagnosis of ADMH.

RESULTS

Most patients (39.28%) were 18–30 years old, and most were female (60.71%). Pigmentation is most common on the face, followed by the upper limbs. No significant difference ($\chi 2 = 5.653$, P= 0.463) was seen between dermatoscopic pigment structures and lichenoid infiltration density on histology. A significant correlation ($\chi 2 = 8.264$, P = 0.016) was identified between epidermal melanization and an enlarged pseudo-reticular pigmentary network. The correlation between exaggerated pseudo-reticular pigmentary network and epidermal thickness was not significant ($\chi 2 = 4.050$, P = 0.132). Significant association between illness duration and melanophage density (r (54) =.862, $p < 0.0001$).

CONCLUSION

The relationship between clinical and dermatoscopic severity and serial dermatoscopy's role in monitoring therapy response and disease progression requires further investigation. The study of illness mechanisms that cause epidermal melanosis and diseases that cause dermal melanosis, including ADMH, is crucial. Future clinical, histological, and dermoscopic studies may help diagnose and prognose ADMH.

Key Words: Dermoscopy, disease progression, Hyperpigmentation, Lichen Planus, Melanosis

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

The global variability of inherent pigmentation is well-documented, with certain skin tones, particularly among Asian and Indian populations, noted to exhibit a higher susceptibility to pigmentation disorders compared to other groups of people. [1,2] Specifically, pigmentary disorders represent a significant issue in India and profoundly affect the psychosocial aspects of quality of life.[3] Facial pigmentation frequently presents in patients visiting the dermatology outpatient department and is a significant concern for those affected. Facial pigmentation could result from multiple factors and is infrequently diagnosed correctly through a comprehensive history and clinical assessment. Various elements, such as genetic and racial influences, significantly contribute to the overall understanding. Melasma, Riehl's melanosis, lichen planus pigmentatosus, nevus of Ota, periorbital hyperpigmentation, and post-inflammatory hyperpigmentation are among the common clinical signs of face pigmentation, among others. [4]

The development of tiny and large pigmented macules or patches is a clinical indicator of a variety of illnesses together referred to as acquired dermal macular hyperpigmentation (ADMH). Without any clinically noticeable inflammatory skin lesions in the past, histopathological analysis shows indications of either resolved or ongoing interface dermatitis together with pigment incontinence. [5,6] The name is intended to include disorders that have been previously recognized in the literature as lichen planus pigmentosus, ashy dermatosis, and Riehl's melanosis/pigmented cosmetic dermatitis[7-9]

Acquired macules of varying sizes are a characteristic of lichen planus pigmentosus, erythema dyschromicumperstans, and ashy dermatosis, exhibiting hyperpigmentation with a grayish hue. [12-14] These conditions are notable for their depth, surpassing that

of epidermal pigmentary disorders like melasma and lentigines. There is a divergence of opinion among authors regarding whether AD, LPP, and EDP represent the same condition or distinct entities. [15-20] Additionally, a comparable condition characterized by smaller macules of acquired hyperpigmentation has been documented, yet the connection with AD, LPP, and EDP remains ambiguous. [21-22]

A non-invasive technique for seeing and diagnosing pigmented skin lesions is dermoscopy. The improved representation of surface and subsurface features makes it easier to spot morphological traits in the lesions that could otherwise go unnoticed. [4]

Dermoscopic observations reflect the fundamental histopathological characteristics. In pigmentary disorders, conditions can exhibit overlapping histopathological characteristics, which may differ depending on the stage of the disease. However, dermoscopy offers specific benefits, because medical professionals are able to examine the whole damaged region, repeat the process when the problem worsens, and modify or confirm the diagnosis.

This establishes dermoscopy as a reliable and significant instrument for assisting in diagnosis, prognosis, and treatment efficacy monitoring. [23]

Its use has expanded lately to cover the diagnosis of a variety of pigmentary and nonpigmentary disorders, having first been used for the identification of skin cancers. There is a dearth of information on thedermatoscopic features of ADMH. [16,24]

In order to describe the dermatoscopic features of ADMH and to establish a link with histological results, this investigation was carried out.

Aim of the study: To assess ADMH's dermatoscopic features and determine how they relate to histopathological results.

Primary objective

To associate dermoscopic findings with histopathological findings in acquired dermal macular hyperpigmented disorders of face.

Secondary objective

To describe dermoscopic features of the acquired dermal macular hyperpigmented disorders of face

REVIEW OF LITERATURE

Disorders related to pigmentation frequently present in dermatological practice. Skin pigmentation represents a significant variable phenotype in humans, with global recognition of skin tone variability. Certain skin types exhibit a greater susceptibility to pigmentary disorders compared to others. The occurrence and psychological effects of these conditions differ according to geographical regions and skin types. Darker-skinned people are more susceptible to pigmentary illnesses because of the contrast between their lower pigment levels and a propensity for post-inflammatory hyperpigmentation. [25]

Identifying Indian skin as “Asian skin” or “skin of color” may not be entirely precise, despite India's clear geographical affiliation with Asia. [26] The population of India exhibits an impressive range of diversity, encompassing over 2,000 distinct ethnic groups. Furthermore, significant differences are present regarding socioeconomic, dietary, and climatic aspects throughout the country. Given this, the variety of Indian skin is evident in both its color and certain distinctive characteristics. [27-30]

Hyperpigmentation leads to disfiguring lesions that can profoundly impact an individual’s psychological and social well-being, leading to a decline in self-esteem, social functioning, and productivity

, particularly in cases of facial hyperpigmentation.[31] Hyperpigmentation refers to the unusual skin darkening, often brought on by an increase in the synthesis of melanin

.[31] This can manifest in the epidermis, dermis, or a combination of both, depending on the location of the irregularity.

Problem statement

"It has been identified as one of the leading skin conditions encountered by dermatologists, accounting for 24.7 million visits related to the management of alterations in skin color." Individuals often experience social and emotional insecurities due to feelings of embarrassment and self-consciousness, leading them to shy away from social events or alter their clothing choices. [32]

Hyperpigmentary disorders rank as the second most prevalent issue among individuals aged 15-30 years, while they emerge as the most common concern for those aged 40-54 years, regardless of gender or skin type. [33]In the Indian population, hyperpigmentation is common and presents serious clinical difficulties. Melasma is more prevalent in sun-exposed areas at higher elevations in India, where UV rays mostly affect those with darker skin tones. [34]

Skin Complexion and Melanogenesis

"Melanin serves as the main pigment responsible for the coloration of the skin. Melanin is synthesized by epidermal melanocytes via the enzymatic oxidation of tyrosine, a process referred to as melanogenesis that occurs within specialized organelles called melanosomes.[35] Following maturation, melanin is conveyed to the adjacent keratinocytes. The epidermal melanin unit refers to the relationship between a single melanocyte and approximately forty keratinocytes. The pigmentation of an organism is determined by the quantity of melanins present, their characteristics, and the balance between eumelanin (brown/black pigment) and pheomelanin (yellow-reddish pigment). Additionally, the method of transfer and processing of melanosomes within keratinocytes plays a crucial role. Notably, the number of melanocytes remains

relatively stable at a specific skin location, regardless of the skin color type. The variation in skin colors is influenced by multiple genes, each possessing numerous alleles, which determine the type and quantity of melanin present. Pheomelanin is considered to have photoreactive properties, whereas eumelanin has been shown to dissipate over 99.9% of absorbed UV and visible rays, functioning as the main form of photoprotection. [36]"

The diversity of skin tone stands out as a significant characteristic among humans. Human skin pigmentation exhibits considerable variability and has primarily evolved to manage the penetration of ultraviolet radiation. [2]Globally, there is a clear relationship between skin pigmentation and the regional distribution of UV radiation. . [37]The majority of dark-skinned people are situated closer to the equator, where UVB radiation exposure is higher. Populations with lighter skin tones tend to be found at greater distances from the tropics and nearer to the poles, areas that experience lower levels of UVB radiation. [38]It is thought that women's lighter skin tone evolved to help with the UVB-induced production of vitamin D3, which is essential for pregnancy and nursing.[2]

The pigmentation of Indian skin exhibits considerable diversity.Hourblin et al. conducted an investigation into various skin characteristics, such as hue, with 1204 female participants from four Indian cities. [39]"The findings indicated that the skin tone within the Indian population varied from whitish to brown, with a gradient from lighter shades in North India to darker shades in South India [Figure 1]. The extensive variation in pigmentation observed within the Indian population may arise from the existence of multiple gene polymorphisms."

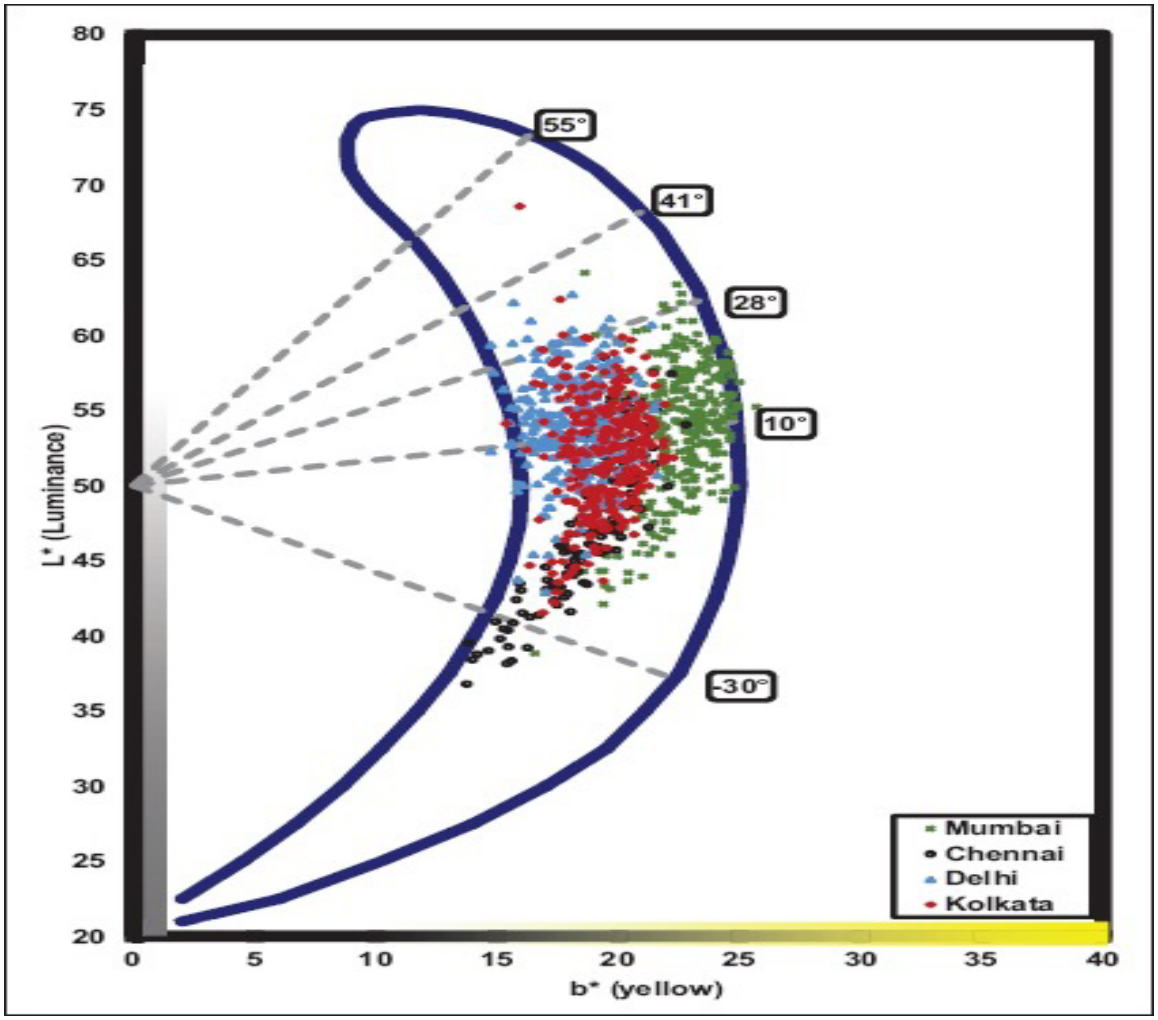


Figure 1: "Indian skin colorimetric classification in the skin color volume projected on the L*/b* plane of the L*a*b* space (CIE 1976). The vertical axis L* is the luminance or lightness of the skin and the horizontal axis b* is the yellow component of theskin.Skin color categories from fair to intense dark and individual typological angles are indicated.[39]"

"Mukherjee et al. examined the allelic frequencies of SNPs in four pigmentation-related genes—*SLC45A2*, *SLC24A5*, *MC1R*, and *TYRP1*—across 749 individuals from 11 sub-populations. The study revealed significant variation in these gene frequencies, suggesting complex and diverse genetic factors influencing pigmentation. These genes are involved in melanin transport and synthesis, with notable effects on skin, hair, and eye color. The findings highlight the role of evolutionary adaptation in pigmentation

diversity, emphasizing the need to consider population-specific genetic differences when studying such traits.[40]"

"Acquired dermal macular hyperpigmentation" (ADMH)

ADMH is an unrelenting condition presenting with hyperpigmentation of various shades, from brown to slate colour to grey shade, without any preceding inflammation. It includes lichen planus pigmentosus, Riehl's melanosis (a type of pigmented contact dermatitis), and ashy dermatosis (erythema dyschromicum perstans).[41]"

Different Disorders of hyperpigmentation

Ashy dermatosis

Ashy dermatosis, initially characterized by Ramirez in 1957, [42] is a condition marked by acquired macular hyperpigmentation that is idiopathic. Because of the ashy blue-grey color of the lesions, the name "ashy or loscenicientos" is used. The face, neck, torso, and upper limbs are the most often affected locations, however any portion of the body might be affected. [43]

Epidemiology

The precise incidence and frequency of Ashy dermatosis remain undetermined. The onset may manifest at any age; however, the average age of occurrence typically falls within the second decade of life. [44,45]

The condition is predominantly observed in Asia and Central and South America, with the majority of documented instances originating from these areas. [44,45]According to Silverberg and colleagues, Hispanics (36%) and Caucasians (52%) made up the bulk of prepubertal individuals with ashy dermatosis. [46]There are two case reports of this disease in Indian youngsters.[47,48] The condition impacts individuals of all genders, though it is observed more frequently in women, [44-46]

Aetiology and Associations

The precise etiology of ashy dermatosis remains unidentified. Nonetheless, there have been reports linking it to the consumption of barium sulfate and ammonium nitrate. HIV infection, whipworm infestation, and hepatitis C virus infection are among the known associated illnesses and infestations. [44-46]

Pathophysiology

It is still unknown what causes erythema dyschromicumperstans, another name for ashy dermatosis. There is evidence linking worm infestations, viral illnesses, and contrast agents. [44-46]

It has been suggested that hyperpigmentation may be the outcome of an immunological reaction to these substances.[49]Furthermore, Numata and his colleagues have suggested that erythema dyschromicumperstans may be caused by a stronger immune response, whereas ashy dermatosis is linked to a weakened immune response. [49]

Clinical Features

The condition typically manifests in young adults during the initial two decades of life. [49] The initial observation reveals a gradual emergence of asymptomatic macules ranging from 0.5 cm to 2 cm, exhibiting diverse shades of gray. [49] The process usually initiates in the trunk and subsequently extends to encompass the face and extremities

The majority of cases seen at our hospital, however, affect the face, neck, and upper trunk significantly, with no involvement seen in the extremities.

The lesions typically exhibit a photo distribution pattern, with a notable sparing of the neck creases. Several authors [44,49] have reported the existence of a raised erythematous border, measuring 1-2 mm in width, that encircles the lesions during the initial phase. It is widely acknowledged by numerous authors [49,50] that the border tends to vanish after a few months. The initial erythematous border may not be evident in every case and is particularly challenging to discern in individuals with Fitzpatrick skin types IV and V.[44] Notably spared are the scalp, nails, mucous membranes, palms, and soles.

Approach to diagnosis

An algorithm developed by Chandran and Kumarasinghe [6] provides a method for diagnosing these conditions, wherein Ashy dermatosis is identified by the presence of grayish-tinged big macules (> 5 cm) or a mix of tiny (< 5 cm) and large macules without an erythematous border.

Dermoscopic features of Ashy dermatosis

The dermoscopic characteristics of Ashy dermatosis are determined by the presence of dots and globules, the colors of the background, and the arrangement of blood vessels. [50] The hue and dimensions of the dots and globules in EDP play a significant role in distinguishing it from LPP. The presence of melanophages and pigment deposits is indicated by the little gray-blue spots and globules on a bluish background that are hallmark dermoscopic features of EDP, as well as inflammatory infiltrate in the deeper dermis. This contrasts with LPP, where larger brownish-gray dots and globules are observed, indicating lichenoid inflammation situated just beneath the epidermis, along with superficially located pigment incontinence. [50]

The investigation conducted by Elmas et al. [51] revealed that the most prevalent type of arrangement is the irregular linear configuration of dots and globules, followed by circular and reticular arrangements. The lesions in EDP exhibit a truncal distribution, characterized by an absence of predilection for perifollicular or periappendageal regions, and do not display an accentuated pseudoreticular pigment network. [52]

Treatment Options

No randomized controlled trials have been conducted to date for the treatment of ashy dermatosis. [53] Currently, there are no treatment options that demonstrate consistent effectiveness for these conditions.

“Topical agents”

“Cosmetics and camouflage creams”

"The application of these creams does not result in any alteration of the lesions; however, they seem to enhance the overall appearance by reducing the contrast with the adjacent unaffected skin."

Narrow band ultraviolet therapy

Tlougan and colleagues [54] support the implementation of narrow band ultraviolet therapy. Nonetheless, the suggestion relies on an isolated instance, lacking details regarding the dosage, treatment duration, and the extent of improvement assessed through an outcome measure.

Tacrolimus ointment

Mahajan and colleagues [55] indicate that in two patients with erythema dyschromicumperstans, the application of topical tacrolimus 0.1% ointment twice daily to the affected areas led to notable improvement after a duration of 3 weeks. It is advisable to conduct controlled trials to determine if the benefit holds statistical significance.

Fractionated laser

The efficacy of non-ablative 1550 nm fractional laser therapy for treating erythema dyschromicumperstans was not established.[56]Furthermore, the treatment led to postinflammatory hyperpigmentation as a consequence of laser burns.

Systemic agents

Clofazimine

Over the past two decades, two investigations were carried out to evaluate the effectiveness of clofazimine in treating erythema dyschromicumperstans. The subjects received a daily dosage of 100mg clofazimine tablets for a duration of 3 months. [57,58] The observed response rates ranged between 66% and 87%. Observed side effects included skin discolourations, corneal changes, and alterations in body fluids, in addition to gastrointestinal intolerance. One potential reason for the limited use of clofazimine may be its tendency to cause increased dyspigmentation relative to the initial condition.

Dapsone

Dapsone, recognized for its antibiotic and anti-inflammatory properties, was utilized by Kontochristopoulos et al..[59] Two patients with erythema dyschromicumperstans have shown a remarkable response to oral dapsone therapy at a dosage of 100mg per day over a period of three months. Following this, Bahadir et al. implemented oral dapsone therapy in a singular instance of erythema dyschromicumperstans, achieving remarkable outcomes. [60] Nonetheless, dapsone carries risks of haemolysis and potentially lethal hypersensitivity reactions, necessitating its use with caution in regulated environments.

"Vitamin A"

"Bhutani administered Vitamin A capsules to 140 patients diagnosed with lichen planus pigmentosus, providing a dosage of 100,000 units daily for a duration of 15 days, followed by a 15-day rest period. The significant number of patients lost to follow-up complicates the ability to determine the precise efficacy of this agent in treating lichen planus pigmentosus. The authors indicate that nine out of 12 patients exhibited a good to excellent response following 10 or more courses of the medication. [61]"

“Lichen planus pigmentosus” (LPP)

It is a chronic dermatological condition characterized by persistent, asymptomatic slaty-gray pigmentation, most commonly affecting the face. The actinic variant is distinguished by symmetric, diffuse pigmentation in sun-exposed areas, particularly in individuals with darker skin tones. However, in rarer cases, LPP may present as macular pigmentation in flexural areas, especially among those with lighter skin tones.[62]

Epidemiology

LPP typically manifests in middle-aged individuals, generally emerging during the third to fourth decades of life, with some studies indicating a marginally higher incidence among females. [62]“The photo-distributed or actinic pattern, which is more prevalent, is a condition found in tropical regions and affects individuals with Type IV to Type V skin types. The majority of the reports originate from India, the Middle East, and South America. The inversus type is uncommon, primarily found among Caucasians.[63]

Etiopathogenesis

Pock et al. proposed that a rapid lichenoid reaction takes place prior to the compensatory increase in keratinocyte proliferation seen in typical LP, leading to a swift change of small raised lesions into flat brown ones.[64] The development of the actinic variant is likely influenced by sun exposure and the use of photosensitizing topical

agents. Mustard oil, which contains allyl-isothiocyanate along with oil of Amla, has been proposed as possible triggering factors due to its photosensitization effect.[65,66]

Clinical Features

LPP presents as a gradual development of pigmentation, lacking any signs of inflammation or prior elevated lesions. It is generally without symptoms, though it may sometimes present with mild itching. The progression of the condition varies, with certain instances demonstrating natural resolution occurring within a timeframe of weeks to months. It can endure for many years in numerous cases. Interestingly, there is no participation of the scalp, mucosa, or nails. The traditional form of LPP is defined by slaty-gray to brownish-black pigmentation that appears symmetrically on areas exposed to light, primarily the face and neck, and subsequently the upper limbs. Instances of more extensive lesions and truncal involvement are uncommon."

While the pigmentation is typically diffuse, various patterns such as reticular, blotchy, perifollicular, annular, and gyrate can also be observed.[66-68] Isolated case reports exist detailing linear unilateral hyperpigmentation in the extremities (Blaschkoid)[69] as well as segmental patterns observed on the trunk.[70] In a study involving Indian patients conducted by Kanwar et al.[66] it was found that a significant majority, 77.4%, exhibited diffuse pigmentation, while smaller percentages displayed reticular (9.7%), blotchy (7.3%), and perifollicular (5.6%) patterns.

Dermatoscopy of LP Pigmentosus

The examination of LPP lesions through dermoscopy has shown pigmentation manifesting in various nonspecific patterns.

Friedman et al.[71] characterized the pigmentation in lesions of LPP as exhibiting diffuse, dotted, and mixed patterns. The diffuse pattern pertains to areas of brown pigmentation that lack a defined structure, likely originating from epidermal pigmentation. The dotted pattern is characterized by fine or coarse blue-gray dots that indicate the presence of dermal melanophages, while the mixed pattern pertains to lesions that exhibit both epidermal and dermal components. The presence of a dotted pattern was associated with a tendency for the pigmentation to remain.[72,73]

Histopathology in Lichen Planus Pigmentosus

The histopathological features of LPP present a continuous spectrum, beginning with pronounced inflammation at the interface in the earliest lesions. This inflammation eventually diminishes, resulting in the distinctive dermal pigmentation observed in more advanced lesions. [74,75] The epidermis typically exhibits atrophy, in contrast to the acanthosis observed in LP.

The inflammatory phase is marked by a dense band of lymphohistiocytic inflammatory infiltrate within the upper dermis, accompanied by notable basal-vacuolar degeneration. Scattered dermal melanophages indicate the presence of some melanin incontinence. The second pattern of burnt-out inflammation presents a minimal superficial perivascular lymphohistiocytic infiltrate, with focal to absent basal-vacuolar degeneration.

Nonetheless, there is significant melanin incontinence accompanied by numerous interstitial and perivascular melanophages. Colloid bodies are frequently observed in

LPP, similar to their presence in LP. CD8+ lymphocytes could represent a significant element within the infiltrate.

Immune deposits are less frequently and consistently observed in LPP compared to classical LP. Approximately 15% of instances exhibit immune deposits. The observed patterns consist of immunoglobulin M (IgM), with immunoglobulin G (IgG) being less common, alongside C3 and fibrinogen deposits in the colloid bodies. Additionally, there are linear deposits of IgM and C3 along the basement membrane zone.[76,77]

Treatment

The progression of the disease remains ambiguous, with certain instances demonstrating spontaneous resolution while others exhibit prolonged pigmentation lasting for years. Furthermore, LPP may exhibit significant clinical and histological similarities with conditions like PCD, making differentiation challenging in standard practice. Due to these challenges, along with the infrequency of the condition, there is limited evidence regarding the effectiveness of the different treatment options. At present, none of the existing treatment alternatives demonstrate reliable outcomes or a definitive advantage over other methods, with the data on effectiveness limited to a small number of case series. Management involves the prevention of aggravating factors, which encompasses photoprotection in the actinic variant. In the inversus variant, it is advisable to implement measures that minimize friction in the body folds, such as reducing weight and avoiding tight clothing. Topical steroids have been widely utilized, yet their effectiveness remains questionable. [68]

An open-label study that was not randomized assessed the efficacy of topical tacrolimus (0.03%) over a duration of 4 months. The findings indicated improvement in seven out of thirteen patients, with noticeable effects beginning at 8 weeks. [68] Earlier reports of poor response to tacrolimus had tried application for shorter durations. Therefore, additional investigations are necessary."

Various systemic treatment approaches have been explored, including the use of systemic corticosteroids and Vitamin A. According to certain authors, the application of dapsone has the potential to impede the advancement of pigmentation. Integrating it with topical tacrolimus could be a viable approach, particularly in instances involving significant pigmentation. [70]

Riehl's melanosis

It is a pigmentation disorder that is acquired and usually impacts areas such as the face, neck, and upper chest, particularly concentrating on the forehead and zygomatic and/or temporal regions. [71] It was first identified and characterized by Riehl. [72]

Definition

Riehl's melanosis was previously referred to as “war dermatosis” [73] due to its disappearance following the conclusion of the war, and subsequently as “melanosis facieifeminae”. [74] In 2018, a worldwide agreement outlined Riehl's melanosis as a condition defined by multiple fine or reticulate, acquired macules of pigmentation with an unclear cause. [75]

Etiology

Since Riehl's initial description of this condition, numerous researchers have examined and documented it. There is a general agreement that Riehl’s melanosis is likely a pigmented contact dermatitis resulting from antigens found in cosmetics and textiles, alongside anecdotal evidence of airborne contact dermatitis related to musk ambrette and various plants.[76] Cosmetic allergens, red and yellow pigments found in textile dyes, chromium hydroxide, aniline and azo dyes, bactericidal agents such as carbanilides and ricinoleic acids, hair dyes, red kumkum, and fragrances have also been identified as contributing factors. In patients from India, sensitization through contact with para-phenylenediamine found in hair dyes represents a significant causative factor. [77] Textile allergens such as optical whiteners, dyes, textile finishes, mercury compounds, formaldehyde, and rubber components are believed to contribute to the development of pigmented contact dermatitis. At times, certain occupational allergens such as coal tar, pitch, asphalt, mineral oil, and chromates have been implicated.[78]

Pathogenesis

Frequent exposure to minimal amounts of allergens found in cosmetics and textiles leads to a type IV cytolytic response, which is marked by vacuolar degeneration of basal cells and pigment incontinence, as opposed to a clear eczematous reaction. Exposure to ultraviolet light may play a role in certain cases, as pigmentation frequently

exhibits photo-localization. Additionally, some of the chemicals involved not only promote melanogenesis but are also recognized as photosensitizers. Photo-patch testing can yield similar pigmentation at the sites of the patch test, thereby reinforcing the influence of ultraviolet rays in the development of the disease.[79]

Epidemiology

The incidence and prevalence of Riehl's melanosis remain unclear. While the majority of reports originate from Japan, there have also been documented cases in Europe, South America, India, and South Africa.[80] Typically, this phenomenon is most evident among ethnic groups and individuals with darker skin tones. A greater predilection seems to be observed among women, with a majority of the patients being young to middle-aged females, although cases have also been reported in males and children. A recent investigation revealed that individuals suspected of having PCD (patch-test positive group) were notably older and had a more extended history of hair color usage.[81]

Clinical Features

Riehl initially characterized the condition as 'the dark brown pigmentation affecting the entire face, with the most notable presence on the forehead and in the zygomatic and temporal areas, and in certain instances, it exhibited a greyish tint. The pigmentation exhibited a more pronounced presence laterally on the face, as opposed to the central area. The extension reached the ears, neck, nape of the neck, and extended onto the scalp over a varying distance.

In comparison to LPP, individuals with PCD tend to exhibit a higher likelihood of experiencing symptomatic hyperpigmentation, and mild superficial scaling may be noticeable. Lesions resulting from cosmetics and hair dye typically begin at the hair margins, presenting as ill-defined hyperpigmented patches. These patches predominantly affect regions exposed to hair dye application.

The areas affected are also contingent upon the specific allergen involved—reactions related to textiles frequently affect the anterior thighs and axillae, while the vault remains unaffected. Nonetheless, these clinical characteristics are not definitive, and it is common to encounter overlapping entities in clinical practice. Individuals might exhibit favorable reactions in patch tests and photo patch tests to cosmetic products or their components.[82]

Dermoscopy of Riehl's melanosis

Dermoscopy has demonstrated its effectiveness as a noninvasive technique for the diagnosis of Riehl's melanosis. The prevalent observations include pseudonetwork patterns characterized by brown or gray dots and globules arranged in arcuate, semiarcuate, or hexagonal formations.[83,84] Telangiectatic vessels, small scales, perifollicular hypopigmented halos, and follicular keratotic plugs are also observable.[85]

Histopathology of Riehl's melanosis

A skin biopsy continues to be the definitive method for diagnosis. The primary histological characteristic is the degeneration of the basal layer, leading to pigment incontinence, as well as the participation of pilosebaceous units, which is further

associated with an increase in melanophages within the dermis. [86] Moreover, the infiltration of lymphocytes, mononuclear cells, and eosinophils leads to perivascular inflammatory cell infiltration, as well as inflammatory cell infiltration in the epidermis and dermis, along with alterations at the interface. The majority of perilesional normal-appearing skin in Riehl melanosis exhibits characteristic histological abnormalities, albeit to a lesser degree. [87]

Treatment

The management of Riehl's melanosis continues to be uncertain. Individuals are required to follow the general guidelines meticulously. [88]

Topical skin-lightening agents

Topical whitening treatments that include hydroquinone, tretinoin, glycolic acid, or azelaic acid have been utilized for the management of Riehl's melanosis. Hydroquinone acts as an inhibitor of the tyrosinase enzyme, thereby blocking the transformation of dopa into melanin. The proposed mechanisms of action include the inhibition of DNA and RNA production, the degradation of melanosomes, and the death of melanocytes.

Clinical findings indicate that a 2% [83] or 4% hydroquinone cream demonstrates a therapeutic effect on Riehl's melanosis.[83,84]

Oral tranexamic acid

Tranexamic acid (TXA) is a synthetic derivative of the amino acid lysine that inhibits the interactions between melanocytes and keratinocytes by reducing the activity of epidermal melanocyte tyrosinase. This is achieved through the inhibition of the plasminogen/plasmin system. TXA is commonly utilized in various pigmentation disorders, particularly melasma, with an effective dosage range of 250 mg/d to 500 mg/d over a treatment duration of 8 to 12 weeks.[85]

Glycyrrhizin compound

For an extended period, glycyrrhizin has been utilized effectively in the treatment of various dermatological conditions, demonstrating pharmacological efficacy similar to corticosteroids while presenting fewer and less severe adverse effects. Previous studies indicate that glycyrrhizin exhibits a range of effects, such as anti-inflammatory, antiviral, anti-allergy, anti-carcinogenesis, and antithrombin properties. The suppression of monocyte movement and the triggering of apoptosis present a credible rationale for the underlying mechanism of glycyrrhizin treatment in Riehl's melanosis.[89]

Chemical peels

“Rani et al. employed a formulation containing 33% glycolic acid and 7% kojic acid in a cohort of six PCD patients, alongside ongoing topical treatment, due to the patients' prior minimal improvement. All patients exhibited noticeable clinical improvement following a total of 15 or 16 treatment sessions, with no adverse effects observed.

Alongside the use of topical demelanizing agents and rigorous sun protection while steering clear of allergy triggers, a chemical peel may be utilized as an adjunctive treatment. [90]"

Materials and Methods

Source of Data: Patients who were clinically diagnosed with ADMH of face attending Dermatology, Venerology and Leprosy OPD

Study duration: The study was conducted in the duration from July 2023 to July 2024

Study design: Hospital based cross-sectional study.

Sample Size: Minimum sample size is 56

Formula used for sample size calculation is,

$$n = \frac{p\,(100 - p)Z}{E}$$

where n is the sample size required, p is the percentage occurrence of a state or condition (proportion or prevalence), E is the percentage maximum error required, Z is the value corresponding to level of confidence required.

In dermatoscopic features, dots were observed in 82.4% cases. Considering similar result at 95% confidence level and 10% maximum error, the sample size is given by,

$$n = \frac{82.4\text{x}\ (100 - 82.4)\ \text{x}\ 1.96}{10}$$

$$n = 55.71242 \approx 56$$

Sampling technique:

Non-Probability Purposive sampling was employed to recruit the patients.

A) All patients with clinically diagnosed cases of ADMH attending KLE'S Dr Prabhakar Kore Hospital and Medical Research Centre, Belgaum who voluntarily gave consent were recruited.

B) Informed consent was obtained from all the patients included in this study.

C) Data was collected by one examiner and recorded in case record proforma.

D) All patients in study underwent a detailed history taking, general physical like height,weight, BMI, systemic, dermatological and histological examination.

E) All participants underwent dermoscopic evaluation.

F) All patients underwent skin biopsy.

G) All participants underwent histological examination.

H) Records were maintained and analyzed statistically and relevant pictures attached.

Inclusion Criteria:

Patients aged 18-70 years who were clinically diagnosed cases of ADMH of face attending skin OPD were included in the study,

Exclusion Criteria

- Non consenting patients
- Patients having concomitant infection or any other lesions affecting the face.
- Patients with bleeding tendencies.

Data collection:

- Data was collected by a single examiner and recorded in case record proforma

- A comprehensive history was gathered, and an exhaustive clinical examination was conducted. All recruited patients underwent a dermatoscopic evaluation to observe specific features, and the findings were meticulously recorded in a predesigned proforma. All supplementary observations recorded were duly documented. A skin biopsy was collected from one of the dermatoscopy sites to provide histopathological correlation of the observed features.
- A hand-held DermLite DL4 dermoscope was utilized for evaluation in the Dermatology department. Dermoscopy was conducted on the bilateral preauricular and frontal areas, covering four specific sites for facial involvement. To assess the neck area, dermatoscopy was conducted on both sides (2 locations). Additional body sites were assessed as necessary. The main structures observed through dermatoscopy were categorized as follows:

  Dots are defined as brown, black, or bluish-grey spherical entities measuring less than 0.1 mm in diameter.

  Globules, on the other hand, are characterized as brown, black, or bluish-grey spherical or ovoid forms exceeding 0.1 mm in diameter.

  Blotches are identified as black pigmented regions that lack structural components.[91,92]
- The initial histopathological examination was conducted to confirm the diagnosis of ADMH and to exclude other possible differential diagnoses such as melasma and amyloidosis, which may clinically resemble conditions categorized under ADMH. Following this, all histopathological specimens were preserved until the study's conclusion, at which point they were evaluated for various characteristics: follicular plugging, degree of epidermal melanization (restricted to basal layer, 1+; 1–3 layers, 2+; >3 layers, 3+),

acanthosis/atrophy, intensity of upper dermal lichenoid infiltrate (mild, 1+; moderate, 2+; severe, 3+), density of dermal melanophages (average count of melanophages per field of 2009, after assessing five such fields), and the depth of dermis affected by dermal melanophages (<2, 2–4, and >4 mm).

Any supplementary findings deemed pertinent were likewise recorded.

Results

Table 1: "Age (in years) distribution of Patients with Acquired Dermal Macular Hyperpigmented Disorders (ADMH)"

Age Group (Years)	Number of Patients (n)	Percentage (%)
18–30	12	21.42%
31–40	22	39.28%
41–50	10	17.85%
51–60	9	16.07%
61–70	3	5.24%
Total	56	100%

The table presents the distribution of patients of ADMH by age group. Age Group of 18–30 years comprised over one-fifth of the total patients, representing a significant proportion of the population. The largest age group of the study was 31–40 years, accounting for nearly 40% of the patients. Patients in the age group of 41 to 50 years accounted for 17.85% of the population. The age group of 51–60 years contributed to 16.07% of the study population older adults aged 61–70 constitute the smallest group, making up just over 5% of the patients. The data highlights that the disease or condition primarily impacts younger populations, with a declining trend in older age groups.

Figure 1: "Bar graph showing Age (in years) distribution of Patients with Acquired Dermal Macular Hyperpigmented Disorders (ADMH)"

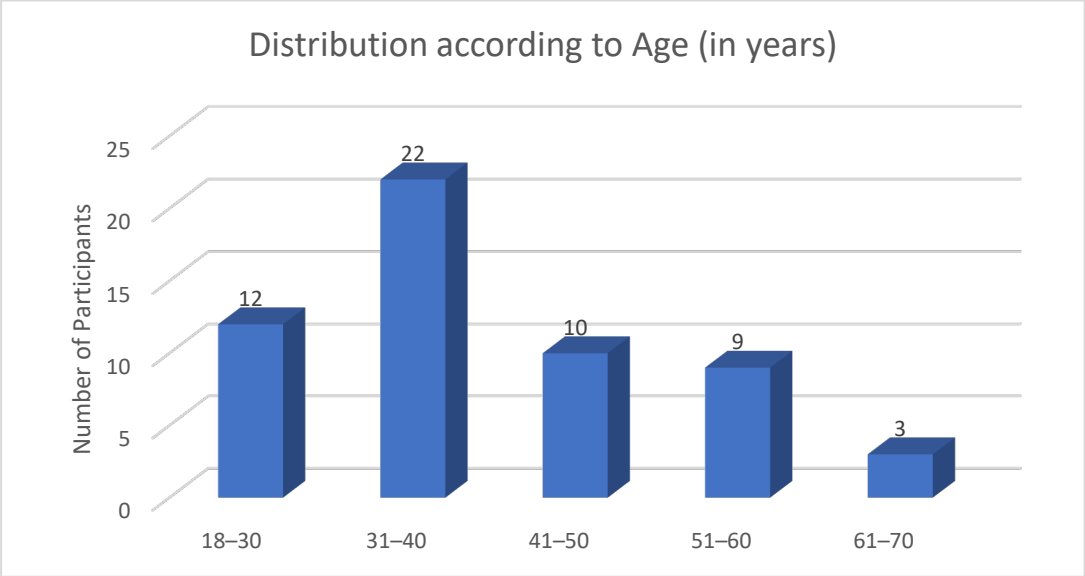


Table 2: "Sex distribution of patients with ADMH"

"Sex"	**"Number of Patients (n)"**	**"Percentage (%)"**
"Male"	"22"	"39.29%"
"Female"	"34"	"60.71%"
"Total"	"56"	"100%"

The data presents an overview of the distribution of patients with ADMH categorized by sex. In the overall population, there were 22 male patients, representing 39.29% of the study population. In the overall population, there were 34 female patients, accounting for 60.71% of the group.

Figure 2: "Pie diagram showing Sex distribution of patients with ADMH"

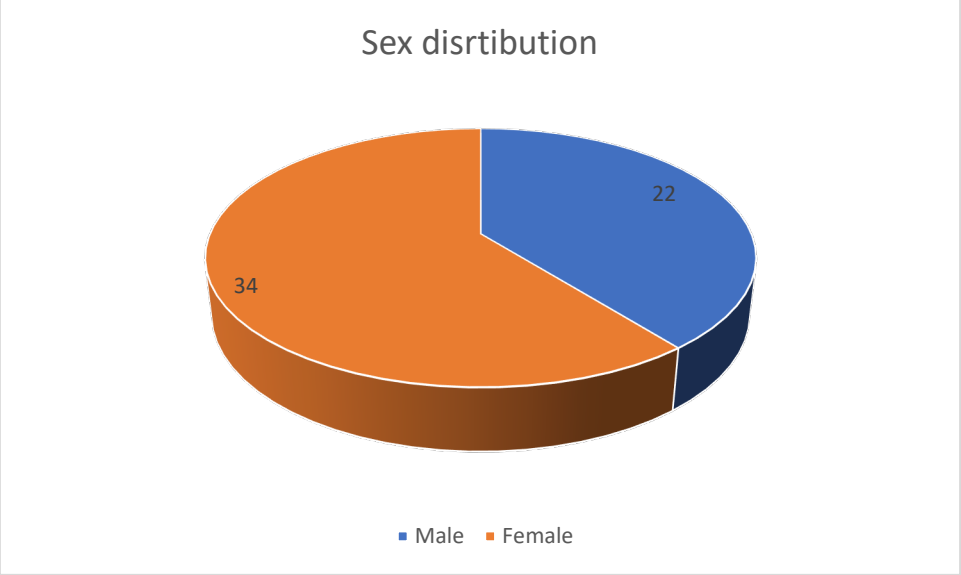


Table 3: Marital distribution of patients with ADMH

Marital Status	Number of Patients (n)	Percentage (%)
Married	40	71.42%
Unmarried	16	28.58%
Total	56	100%

The data provides a comprehensive analysis of the distribution of patients with ADMH, classified according to their marital status. Among the entire cohort, 40 individuals were married, accounting for 71.42% of the total population under investigation. A total of 16 unmarried patients were identified within the overall population, representing 28.58% of the group.

Figure 3: Pie diagram showing marital distribution of patients with ADMH

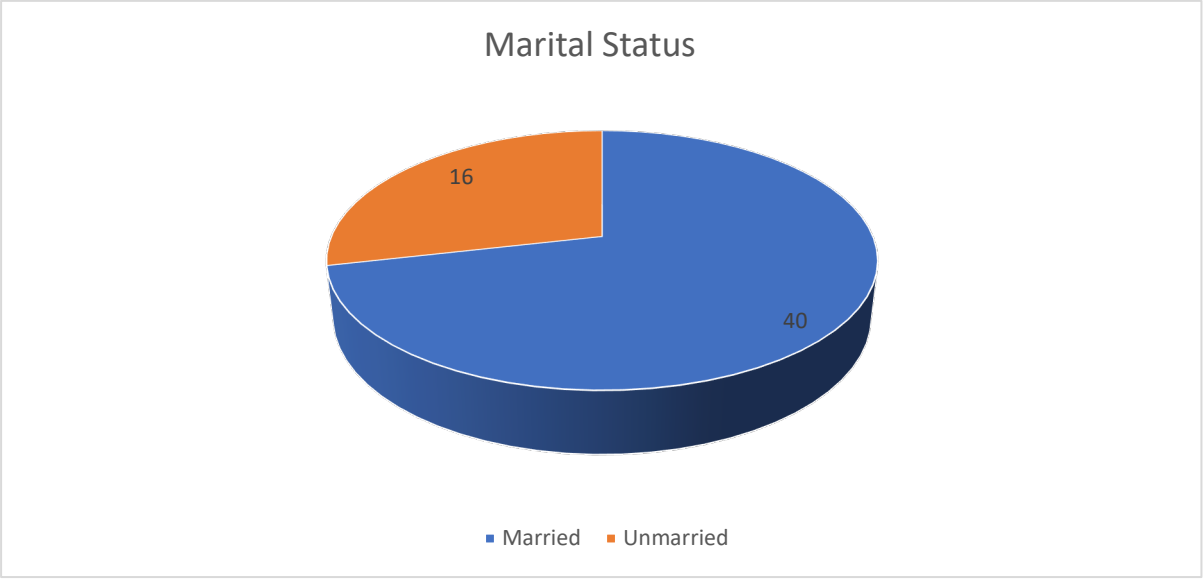


Table 4: "Distribution of patients with ADMH according to type of residence"

Type of residence	Number of Patients (n)	Percentage (%)
Urban	42	75.00%
Rural	14	25.00%
Total	56	100%

The data provides analysis of the distribution of patients with ADMH, categorized by their kind of place of residence. Of the whole cohort, 42 people resided in urban areas, representing 75% of the overall population under examination. A total of 14 patients from rural areas were found among the study population, constituting 28.58% of the group.

Figure 4: "Pie diagram showing distribution of patients with ADMH according to type of residence"

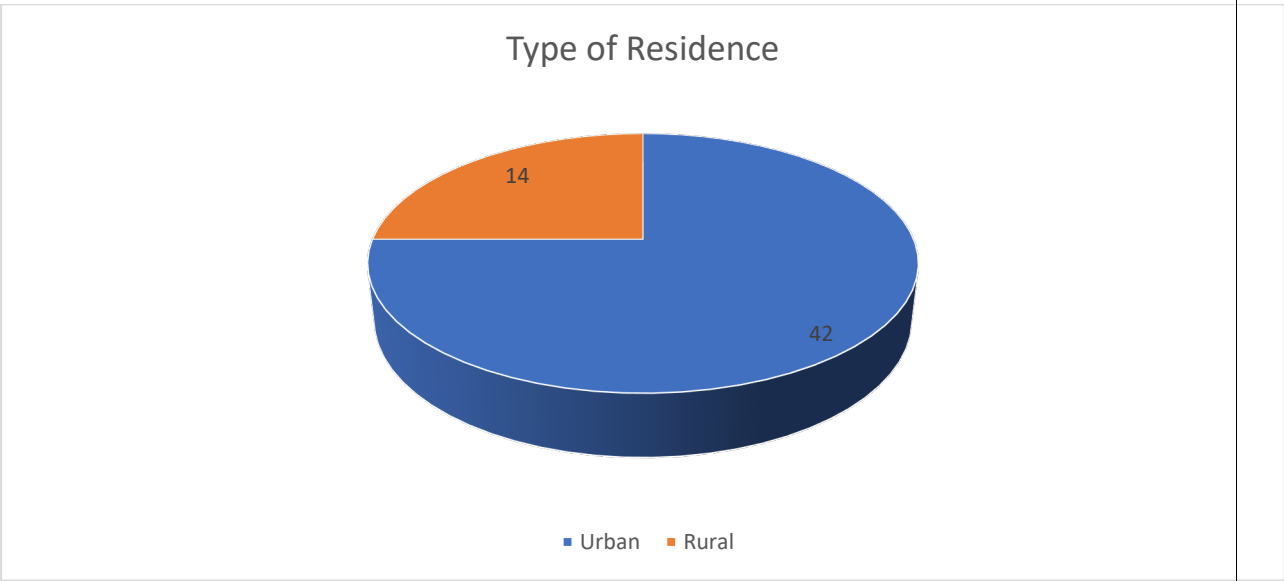


Table 5: Distribution of patients with ADMH according to employment status

Employment status	**"Number of Patients (n)"**	**"Percentage(%)"**
Employed	35	62.50%
Unemployed	21	37.50%
Total	56	100%

The analysis conveys a detailed examination of the distribution of patients diagnosed with ADMH, organized according to their employment status. Among the entire group, 35 individuals were in employment, accounting for 62.50% of the total population being studied. A total of 21 patients were identified as unemployed within the study population, representing 37.50% of the group.

Figure 5: "Pie diagram showing distribution of patients with ADMH according to employment status"

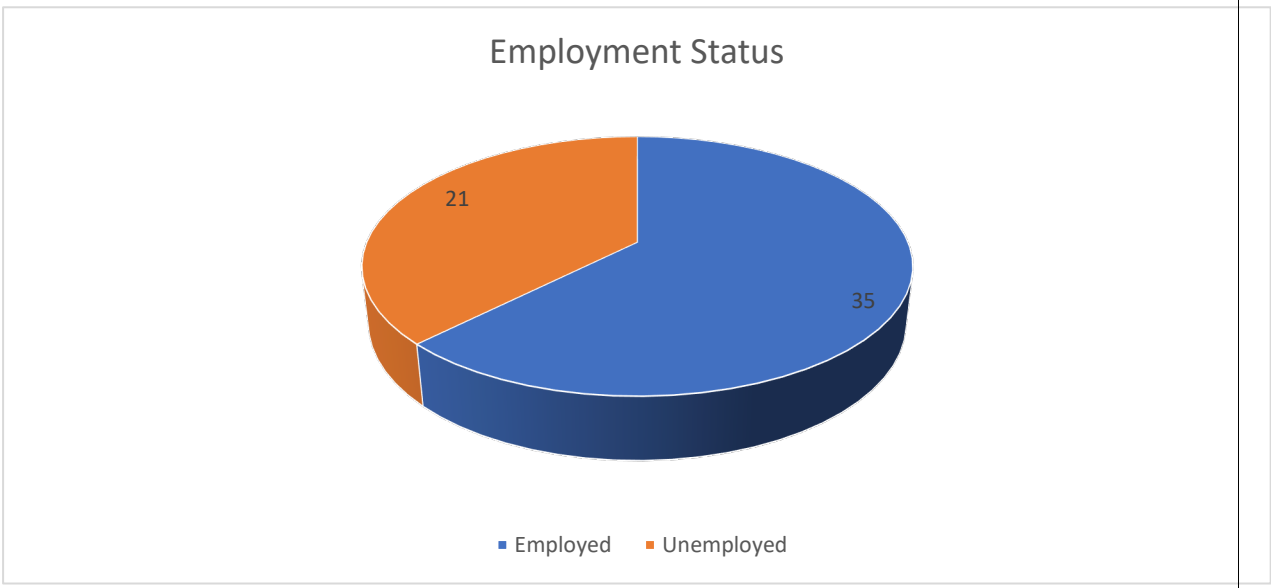


Table 6: Location of Pigmentation in patients with ADMH in the Study Population

"Location of Pigmentation"	"Number of Patients (n)*"	"Percentage (%)"
Face	56	100%
Upper Limb	40	71.42%
Upper Trunk	16	28.57%
Lower Limbs	9	16.07%
Neck	12	21.42%
Abdomen	4	7.14%

***All are not mutually exclusive**

The table provides a detailed breakdown of the locations of pigmentation among patients of ADMH in the study population. The findings are summarized as follows: All patients exhibited pigmentation on the face (100%), making it the most universally affected site. A majority (71.42%) of patients also displayed pigmentation on their upper limbs. Pigmentation on the upper trunk was observed in about one-third of patients (28.57%), making it a moderately affected area. Pigmentation on the lower limbs was less frequent, affecting 16.07% of patients. The neck was affected in just over one-fifth (21.42%) of the patients, showing moderate involvement compared to other regions. The abdomen was the least affected area, with pigmentation present in only a small fraction (7.14%) of patients.

"The data highlights a clear pattern in the distribution of pigmentation, with the face being the most frequently and universally involved site, followed by the upper limbs."

Figure 6: "Bar diagram showing distribution of patients with ADMH according to Location of Pigmentation"

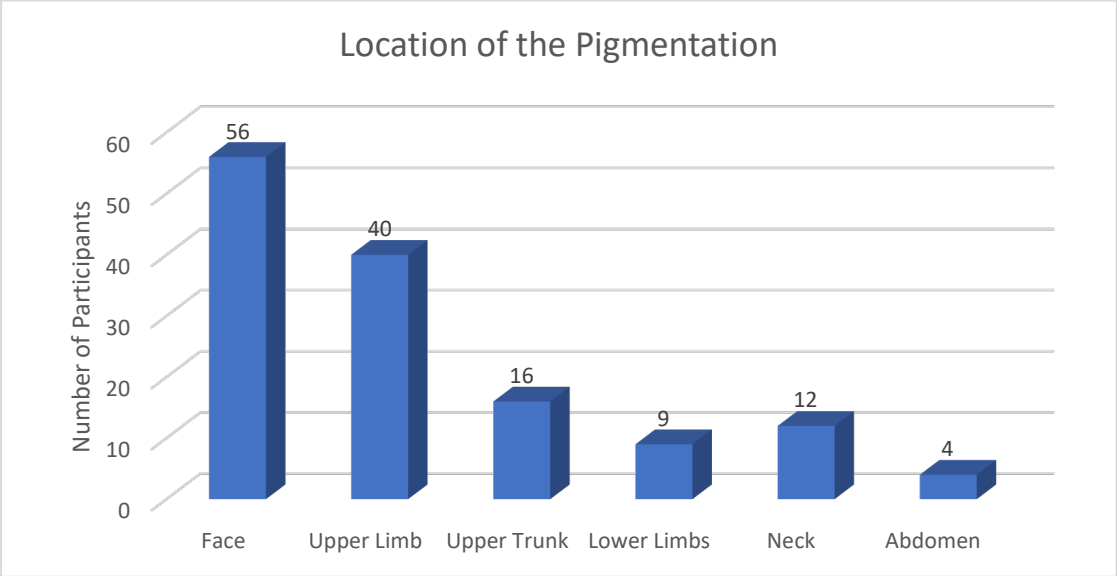


Table 7: "Distribution of Pigmentation in patients with ADMH in the Study Population"

Distribution of Pigmentation	Number of Patients (n)	Percentage (%)
Centro facial	5	8.92%
Malar	1	1.78%
Perioral	1	1.78%
Periorbital	2	3.56%
Forehead	6	10.71%
Malar, retro auricular	5	8.97%
Forehead, retro auricular	14	25.00%
Forehead, malar, retro auricular	22	39.28%
Total	56	100%

The distribution of pigmentation in patients with ADMH was analyzed. Among the 56 patients, the most common pattern observed was pigmentation on the forehead, malar, and retroauricular regions, affecting 22 individuals (39.28%). This was followed by pigmentation on the forehead and retroauricular areas in 14 patients (25%). Pigmentation limited to the forehead was present in 6 patients (10.71%), while the centrofacial and malar-retroauricular patterns were each observed in 5 patients (8.92% and 8.97%, respectively). Pigmentation in the periorbital region was seen in 2 patients (3.56%), whereas perioral and isolated malar pigmentation were the least common, affecting only 1 patient each (1.78%).

Figure 7: "Bar diagram showing distribution of patients with ADMH according to Distribution of Pigmentation"

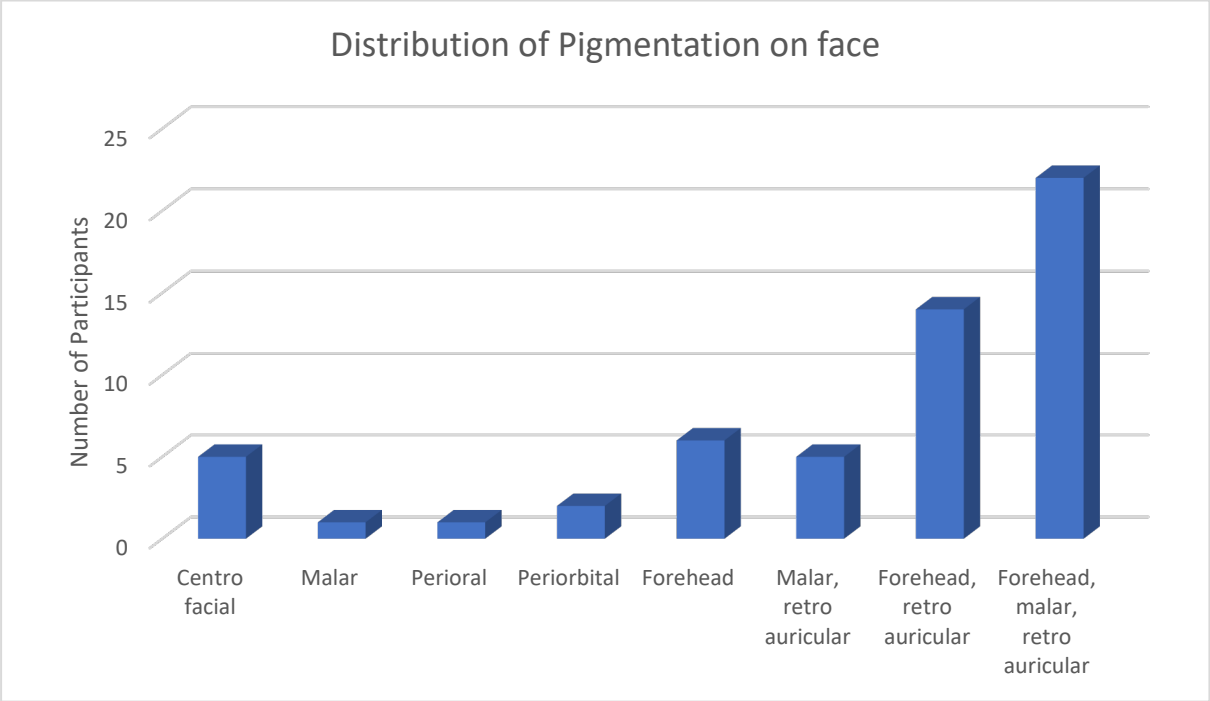


Table 8: Distribution of ADMH patients according to type of color

Type of color	Number of Patients (n)	Percentage (%)
Violaceous	10	17.85%
Greyish black	40	71.42%
Brown	6	28.58%
Total	56	100%

The table presents the distribution of pigmentation types based on color among patients of ADMH. Violaceous pigmentation, characterized by a purplish tone, is observed in 10 patients (17.85%). This color may indicate underlying vascular involvement, inflammation, or specific conditions associated with dermal changes. Greyish-black pigmentation is the most common type, affecting nearly three-quarters (71.42%) of the patients. This darker tone might suggest chronicity, deeper dermal melanin deposition, or environmental factors like prolonged sun exposure or drug reactions. Brown pigmentation, associated with superficial melanin deposition or milder hyperpigmentation, is the least common type (28.58%).

Figure 8: Bar diagram showing distribution of patients with ADMH according to Type of colour

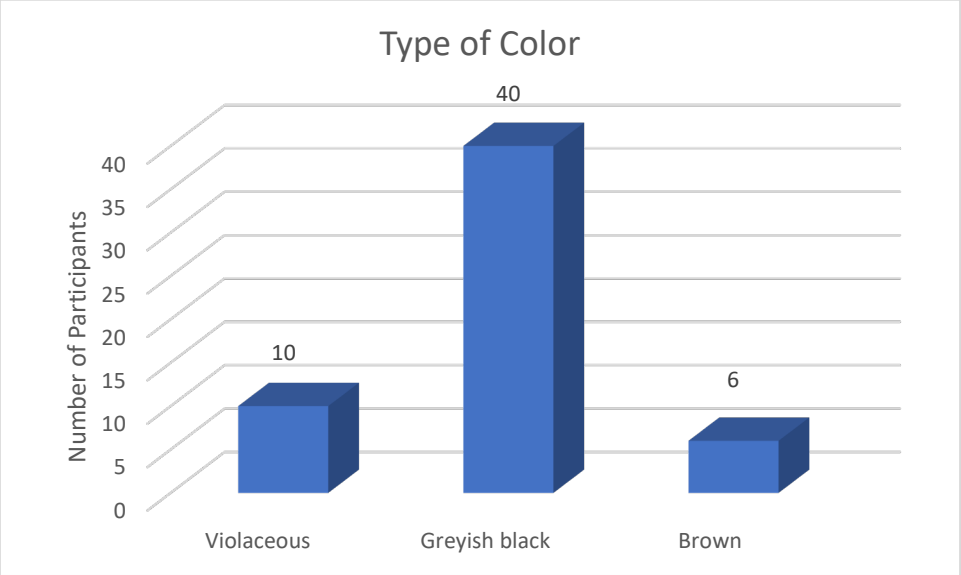


Table 9: Pre-Existing comorbidities in ADMH Patients

Pre-Existing comorbidities	Number of Patients (n)	Percentage (%)
Diabetes Mellitus (DM)	9	16.07%
Hypertension	7	12.50%
Hypothyroidism	5	8.92%

The table summarizes the pre-existing comorbidities among patients of ADMH in the study population. DM is the most common (16.07%) comorbidity, affecting the patients. This is significant as diabetes is often associated with skin changes and conditions, including hyperpigmentation, delayed healing, and susceptibility to infections Hypertension is the second most frequent comorbidity (12.50%). Chronic hypertension can contribute to vascular changes, which might exacerbate certain skin conditions or influence pigmentation patterns. Hypothyroidism is the least common comorbidity (8.92%). This is notable, as hypothyroidism is often linked to skin dryness, pallor, or pigmentation changes, possibly contributing to the observed skin manifestations.

Figure 9: Bar graph showing Pre-Existing comorbidities in ADMH Patients

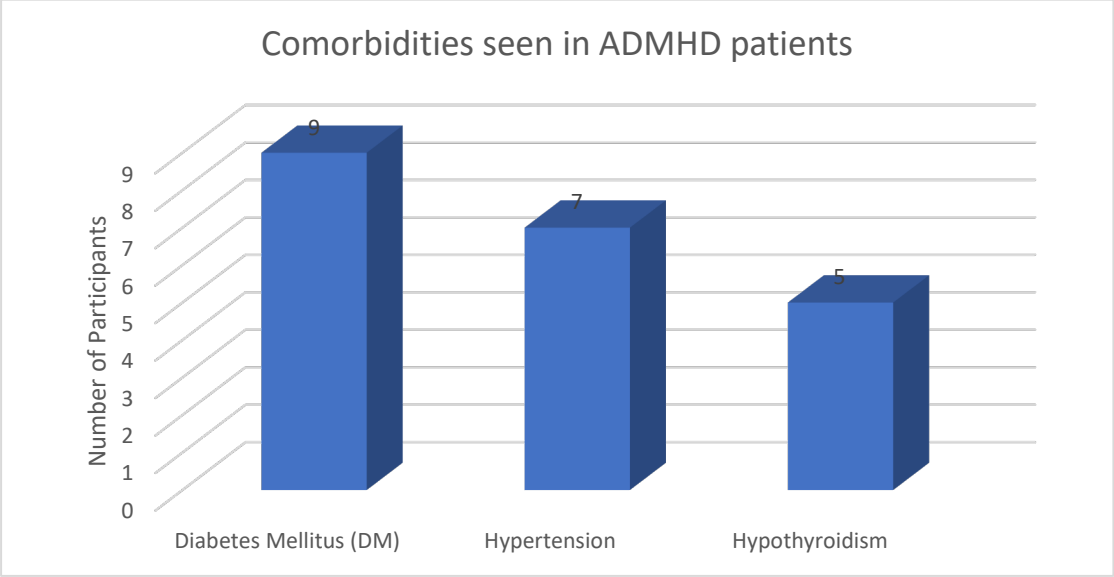


Table 10: Distribution of Etiology of ADMH Patients in the Study Population

Etiology	Number of Patients (n)	Percentage (%)
LPP (Lichen Planus Pigmentosus)	31	55.35%
Riehl's Melanosis	13	23.21%
Ashy Dermatosis	12	21.44%
Total	56	100%

The table presents the distribution of ADMH cases by etiology. Lichen Planus Pigmentosus (LPP) emerged as the predominant cause, identified in 31 patients (55.35%). Riehl's Melanosis was identified in 13 patients (23.21%), while Ashy Dermatosis was present in 12 patients (21.44%).

Figure 10:Bar graph showing Distribution of Etiology of ADMH Patients in the Study Population

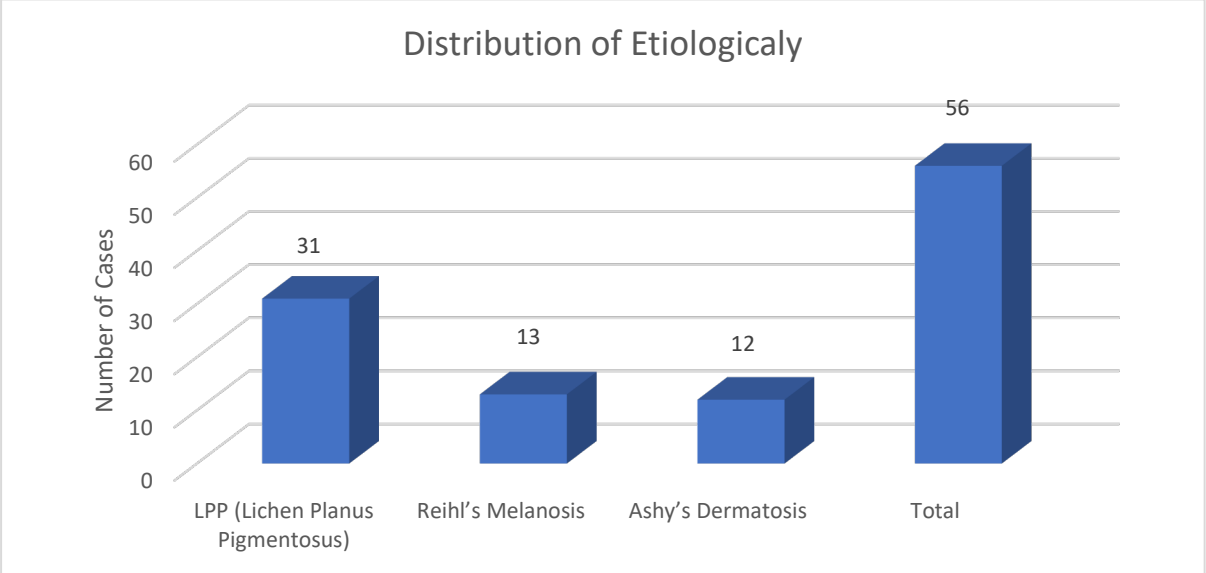


Table 11: "Distribution of ADMH Patients according to the duration of the symptoms (in months) in Study Population"

"Duration of the symptoms (in months)"	"Number of Patients (n)"	"Percentage (%)"
≤ 12 months	28	50.00%
12 – 24 months	4	7.15%
≥ 24 months	24	42.85%
Total	56	100%

The table provides information on the duration of symptoms in patients with ADMH. Half of the patients (50%) reported symptom durations of one year or less, indicating a relatively acute or recent onset. Only a small fraction of patients (7.15%) fell into the intermediate duration range. A significant proportion of patients (42.85%) reported symptoms lasting more than two years, reflecting chronic or long-standing cases.

Figure 11: "Bar graph showing : Distribution of ADMH Patients according to the duration of the symptoms (in months) in Study Population"

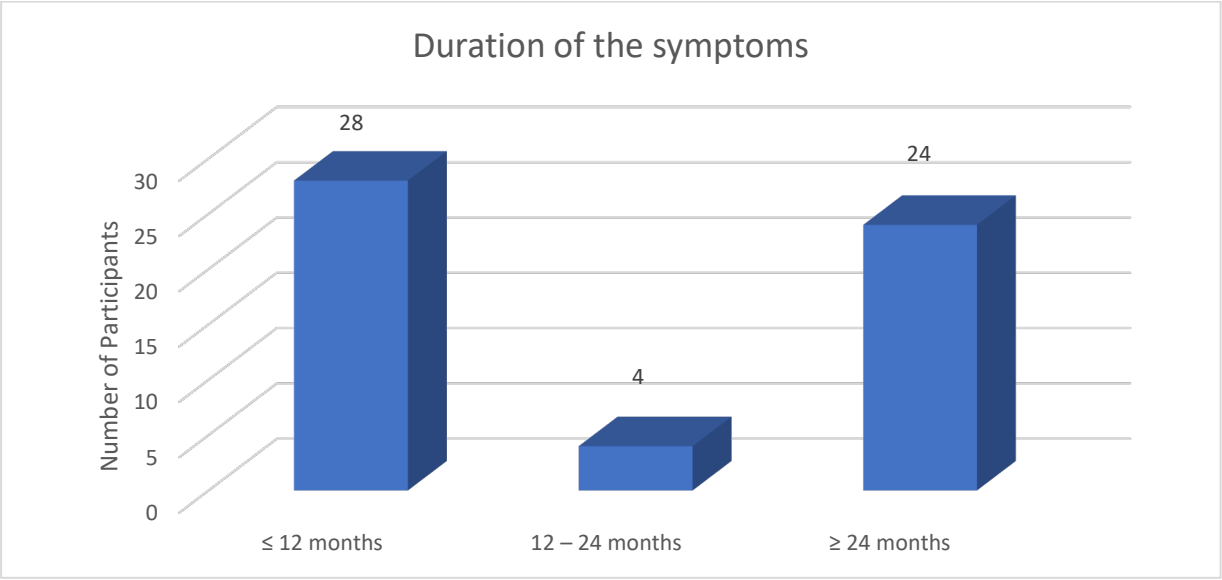


Table 12: Distribution of ADMH Patients according to the Dermatoscopic structures

Dermatoscopic structures	Number of Patients* (n)	Percentage (%)
Dots	35	62.50%
Globules	22	39.28%
Blotches	18	32.14%
Telangiectasia	12	21.42%

*All are not mutually exclusive

The table provides insights into the dermatoscopic structures observed among patients of ADMH, categorized as dots, globules, diffuse patterns, and telangiectasia. Dots (62.5%) are the most common dermatoscopic structure Globules, observed in nearly 40% of patients, typically represent clusters of melanophages or deeper melanin deposits in the dermis. Blotches patterns are noted in about one-third (32.14%) of patients. Telangiectasia, characterized by visible dilated blood vessels, is the least common structure, present in just 21.42% of patients

Figure 12: Bar graph showing Distribution according to dermatoscopic structures in the Study Population

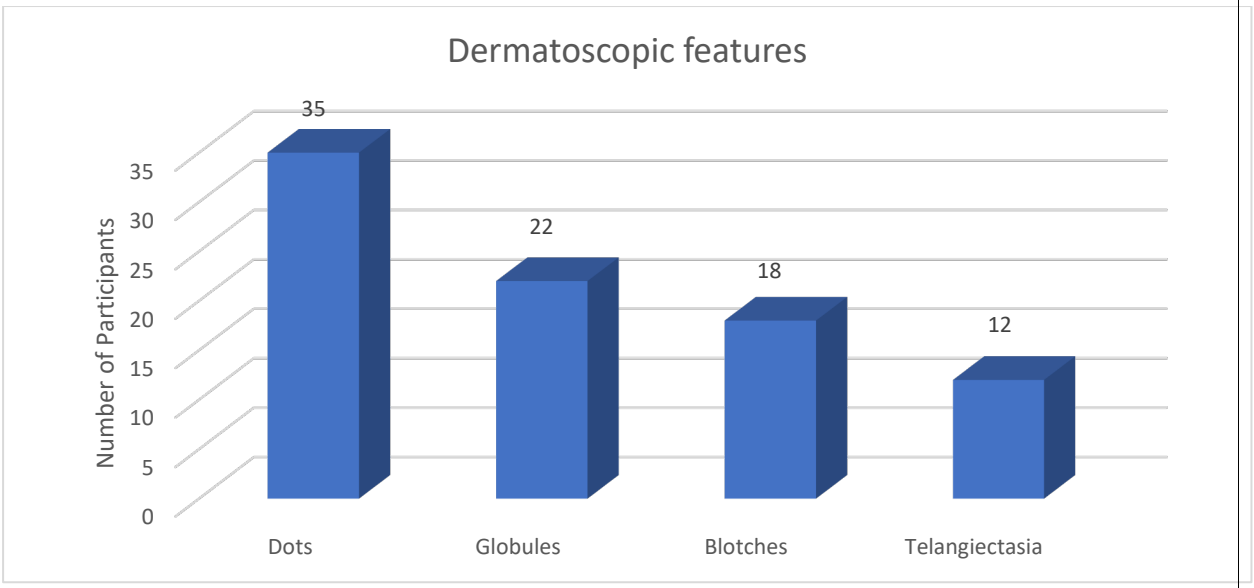


Table 13: Distribution of ADMH Patients according to the Global Features

Global Features	Number of Patients* (n)	Percentage (%)
Sparing of Eccrine opening	53	94.64%
Sparing ofHair follicle openings	51	91.07%
Sparing of Skin creases	20	35.71%

*All are not mutually exclusive

The table summarizes the global dermatoscopic features observed in patientsof ADMH. Sparing of eccrine openings was the most common feature (94.64%), observed in nearly all patients. This phenomenon occurs when pigmentation does not extend over the sweat gland ducts, leaving them visibly unaffected. Sparing of Hair Follicle Openings was present in 91.07% of patients, reflecting the selective involvement of interfollicular skin in pigmentation disorders. Sparing of skin creases was the least common feature (35.71%), observed in approximately one-third of patients.

Figure 13: Bar graph showing Distribution of ADMH Patients according to the Global Features

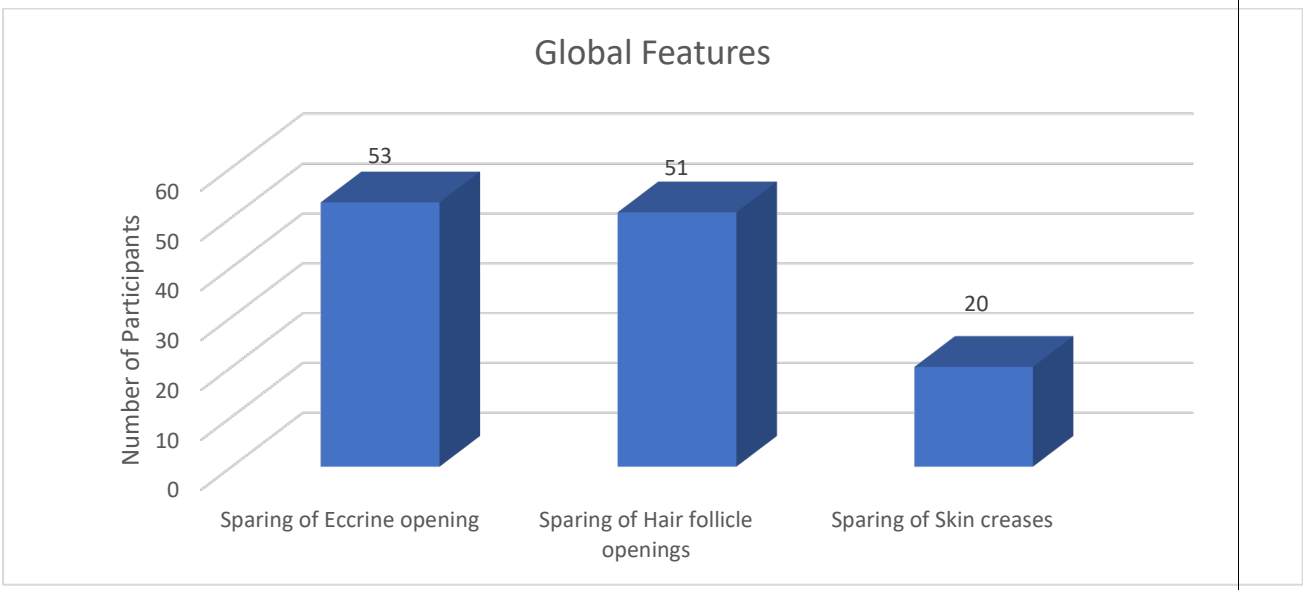


Table 14: Distribution of ADMH Patients according to the Arrangement of pigment structures

Arrangement of pigment structures	Number of Patients (n)	Percentage (%)
Chinese letter pattern	31	55.35%
Diffuse	14	25.00%
Reticular arrangement	11	19.65%
Total	56	100%

The table describes the arrangement of pigment structures observed in patients of ADMH. The data reveals that the Chinese letter pattern is the most common dermatoscopic arrangement (55.35%), particularly associated with melasma and conditions involving irregular pigment deposition. The diffuse pattern (25%) reflects generalized pigmentation, while the reticular arrangement (19.65%) indicates chronic, dermal pigmentation disorders. Dermatoscopic evaluation remains a key tool in managing pigmentation disorders by guiding accurate diagnoses and monitoring therapeutic efficacy.

Figure 14: Bar graph showing Distribution of ADMH Patients according to the pigment structures.

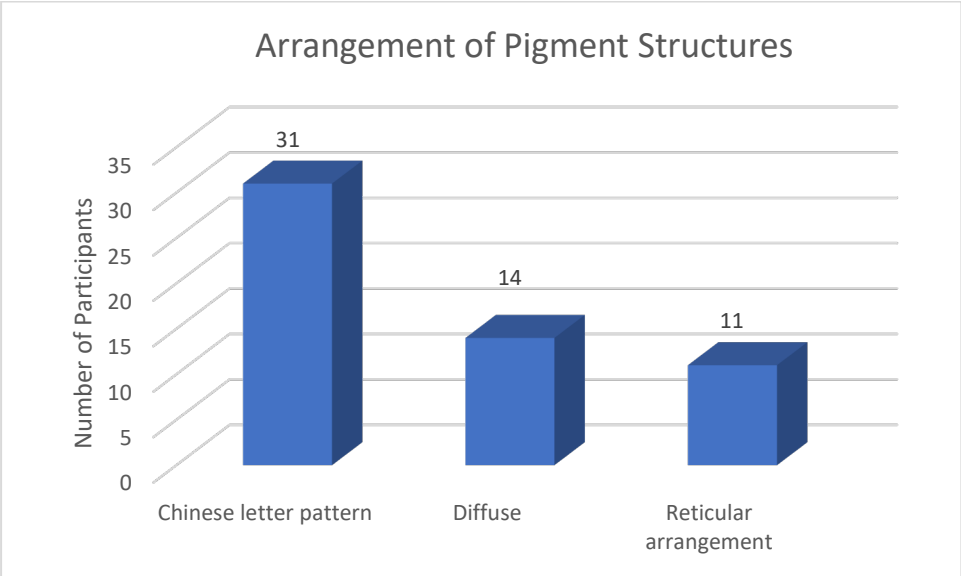


Table 15: Distribution of ADMH Patients according to other global features

Other global features	Number of Patients (n)	Percentage (%)
"Exaggeration of the normal pseudoreticularpigmentary network"	36	64.28%
"Owl's eye appearance"	8	14.28%

"The table highlights distribution of two global dermatoscopic features observed in patients of ADMH: exaggeration of the normal pseudoreticularpigmentary network and the owl's eye appearance. Exaggeration of the Normal PseudoreticularPigmentary Network was observed in 64.28% of patients. The pseudoreticularpigmentary network represents the natural pigmentary pattern of the skin, which becomes exaggerated in certain pigmentation disorders. The owl's eye appearance was present in 14.28% of cases, refers to circular or oval pigmented structures resembling an owl's eyes. These dermatoscopic patterns provide critical diagnostic and prognostic information, guiding tailored treatment strategies and improving patient outcomes."

Figure 15: Bar graph showing Distribution of ADMH Patients according to other global features

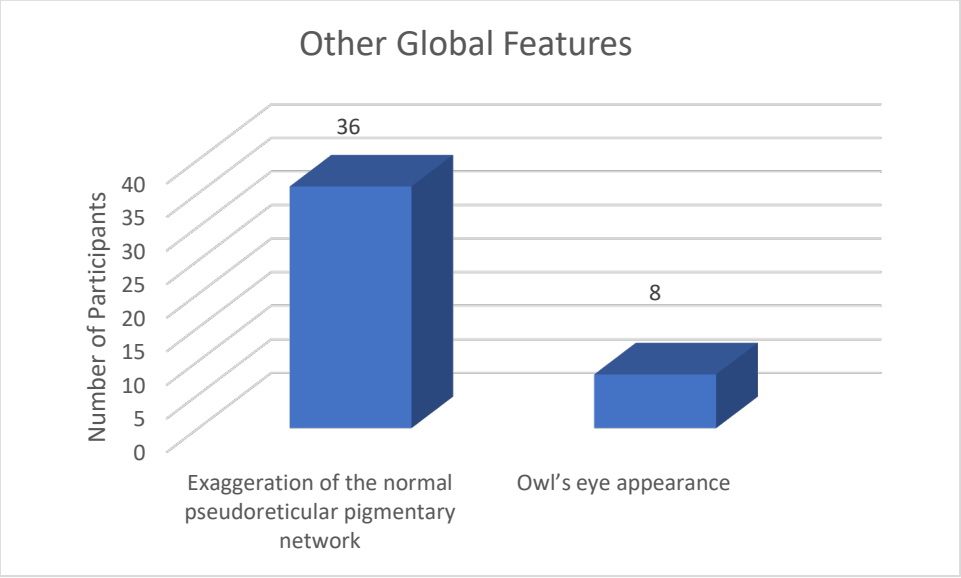


Table 16: Distribution of ADMH Patients according to Epidermal melanization

Epidermal melanization	Number of Patients (n)	Percentage (%)
Basal Layer	9	16.07%
1 - 3	36	64.28%
>3	11	19.64%
Total	56	100%

The table provides data on the epidermal melanization levels in patients of ADMH, categorized based on the extent of pigmentation observed across specific epidermal layers. In 16.07% of patients, melanization was confined to the basal layer of the epidermis. This indicates pigmentation limited to the innermost layer of the epidermis.The majority of patients (64.28%) exhibited melanization extending through 1 to 3 layers of the epidermis. This reflects moderate pigmentation, suggesting that the pigment has spread beyond the basal layer but has not yet reached the full thickness of the epidermis.In 19.64% of cases, melanization extended beyond 3 layers of the epidermis, indicating severe or advanced pigmentation disorders.

Figure 16: Bar graph showing distribution of ADMH according to Epidermal melanization

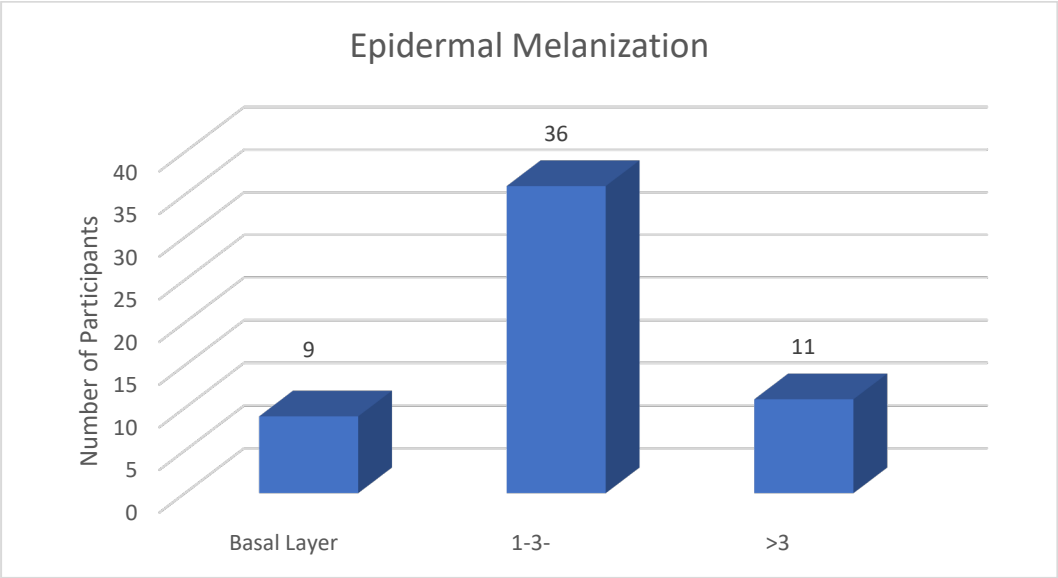


Table 17: Distribution of ADMH Patients according to Epidermal thickness

Epidermal thickness	Number of Patients (n)	Percentage (%)
Normal	14	25.00%
Acanthosis	25	44.64%
Atrophy	17	30.36%
Total	56	100%

The data provides insights into the variations in epidermal thickness among patients of ADMH. The study reveals that acanthosis (44.64%) was the most common alteration in epidermal thickness among patients with pigmentation disorders, indicating chronic and persistent conditions. Atrophy (30.36%) was also significant, reflecting advanced or degenerative cases, while normal thickness was observed in 25% of the patients of ADMH. These findings highlight the importance of assessing epidermal thickness to guide treatment decisions and monitor disease progression.

Figure 17: Bar graph showing distribution of ADMH according to Epidermal thickness

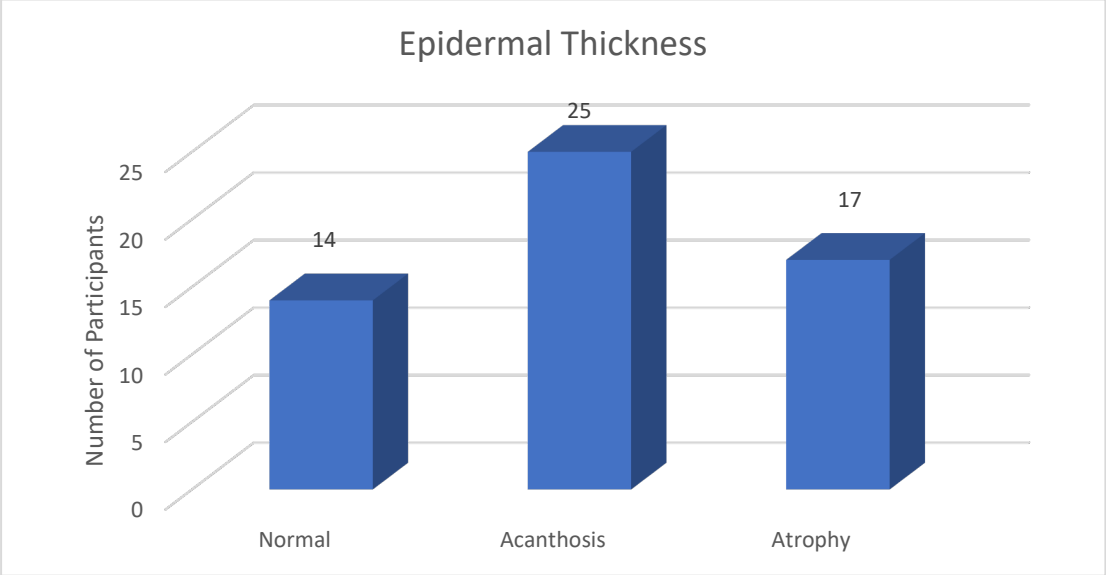


Table 18: Distribution of ADMH Patients according to Upper dermal lichenoid infiltrate

Upper dermal lichenoid infiltrate	Number of Patients (n)	Percentage (%)
None	9	16.07%
Mild	35	62.50%
Moderate	8	14.28%
Severe	4	7.60%
Total	56	100%

The table provides data on the degree of upper dermal lichenoid infiltrate observed in patients of ADMH In 16.07% of patients, no lichenoid infiltrate was detected. The majority of patients (62.50%) exhibited mild infiltrate, reflecting low-grade dermal inflammation. Moderate lichenoid infiltrate was observed in 14.28% of patients, indicating a more significant inflammatory response in the dermis. Severe infiltrate was the least common, seen in only 7.60% of patients.

Figure 18: Bar graph showing distribution of ADMH according to Upper dermal lichenoid infiltrate

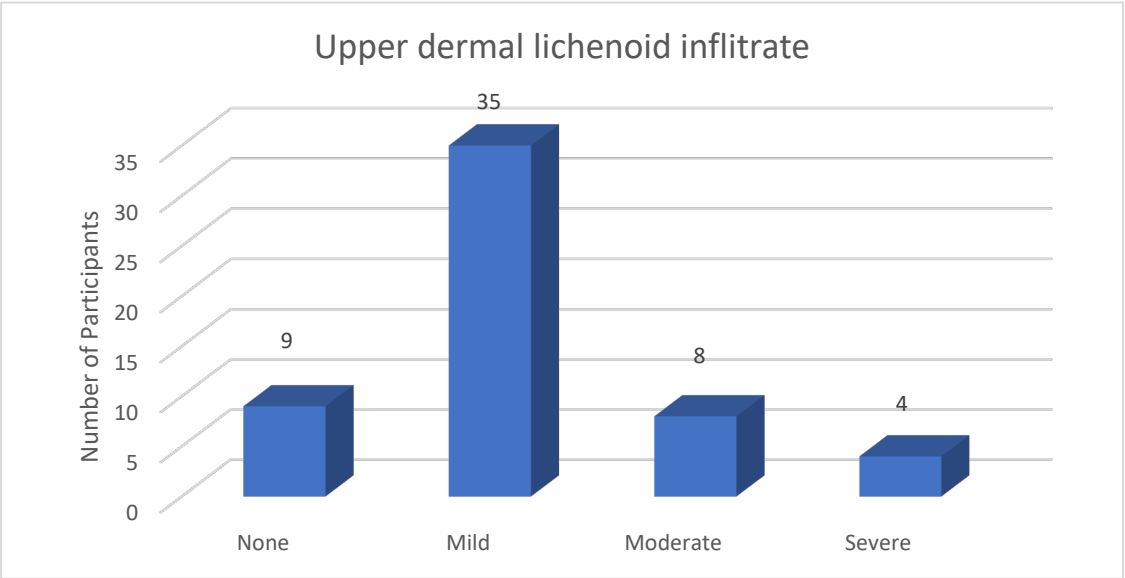


Table 19: Distribution of ADMH Patients according to density of melanophages (per high power field 400×)

Density of melanophages (per high power field 400×)	Number of Patients (n)	Percentage (%)
≤10	21	37.50%
11-15	18	32.14%
>15	17	30.36%
Total	56	100%

The data categorizes the density of melanophages (pigment-laden macrophages) per high-power field (400× magnification) among ADMH patients. The data revealed a distribution of melanophage density where ≤10 melanophages per field (37.50%) was the most common, followed closely by 11–15 (32.14%) and >15 (30.36%). These findings suggest a wide spectrum of pigment deposition among patients, ranging from mild to severe.

Figure 19: Bar graph showing Distribution Patients according to density of melanophages

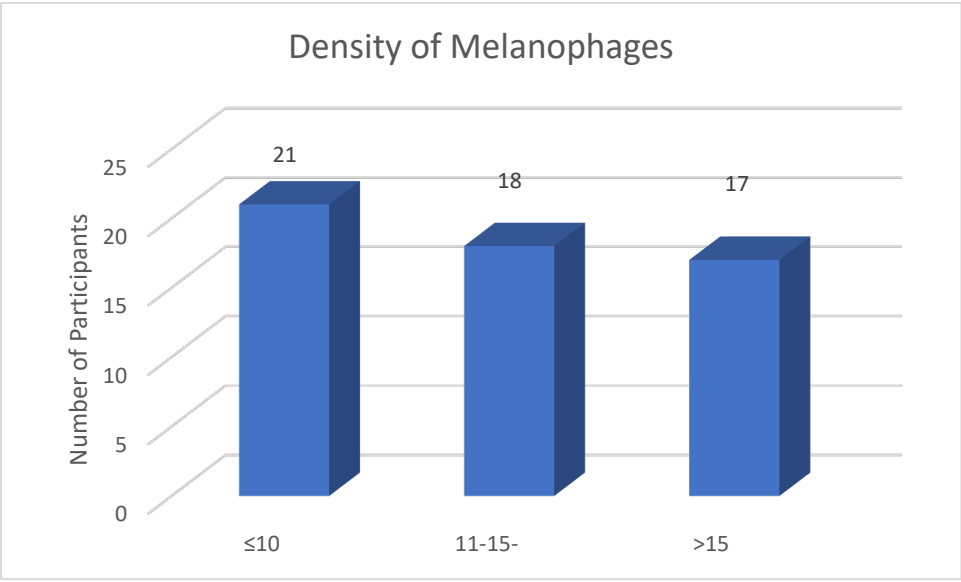


Table 20: Distribution of ADMH Patients according to severity of inflammatory infiltrate

Severity of inflammatory infiltrate	"Number of Patients (n)"	"Percentage (%)"
"Grade 1"	14	25.00%
"Grade 2"	38	67.85%
"Grade 3"	4	7.15%
Total	56	100%

The analysis focused on the distribution of ADMH patients according to the severity of inflammatory infiltrate. Out of the 56 patients, a significant portion (67.85%) displayed Grade 2 inflammatory infiltrate, which corresponds to 38 individuals. Infiltrate classified as Grade 1 was noted in 14 patients, accounting for 25% of all cases examined. The least frequent severity observed was Grade 3, noted in merely 4 patients, accounting for 7.15% of the study cohort.

Figure 20: Bar graph showing distribution of ADMH according to severity of inflammatory infiltrate

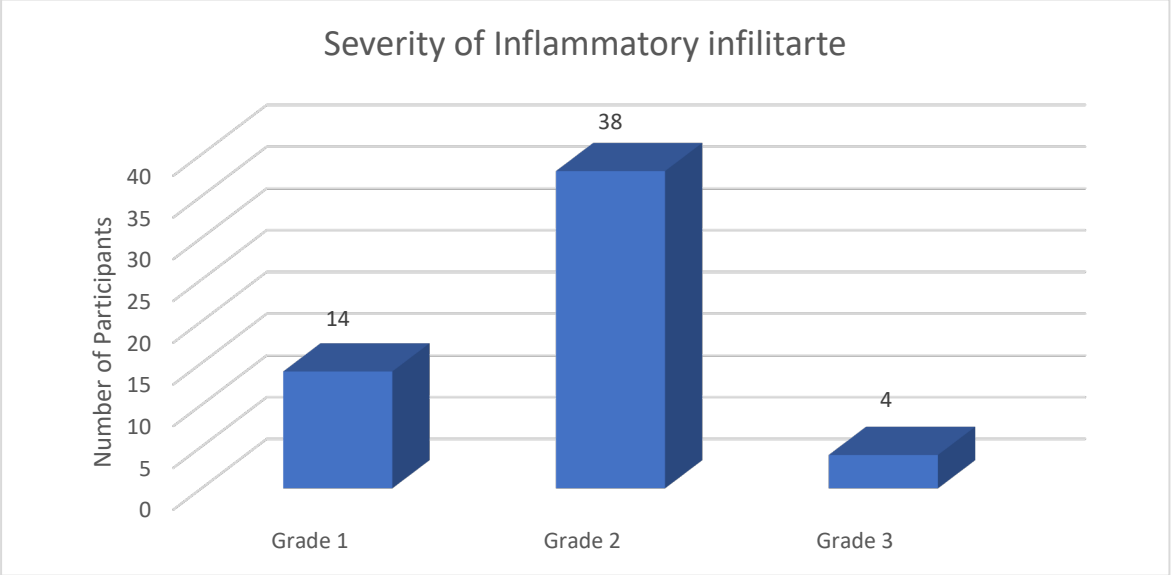


Table 21: Distribution of ADMH patients according to presence of rete ridges

Rete ridges	**"Number of Patients (n)"**	**"Percentage (%)"**
"Present"	25	44.64%
"Absent"	31	55.36%
"Total"	56	100%

The distribution of ADMH patients based on the presence of rete ridges was analyzed. Among the total patients, 25 individuals (44.64%) exhibited rete ridges, while 31 patients (55.36%) did not. The absence of rete ridges was observed more frequently than their presence in the study population.

Figure 21: Pie diagram showing distribution of ADMH according to presence of rete ridges

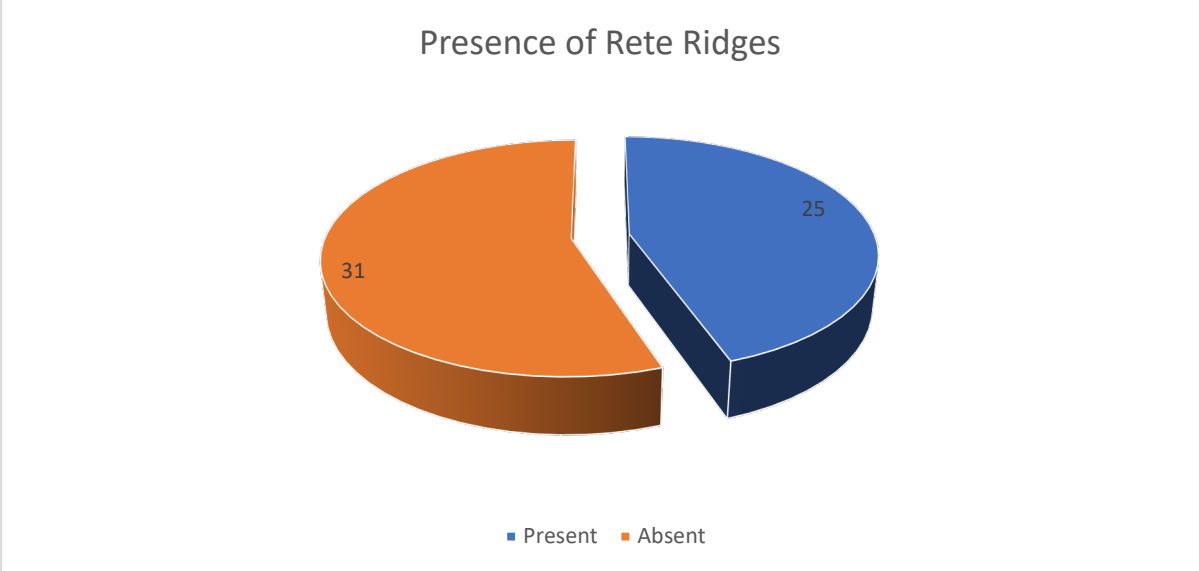


Table 22: Association of dermatoscopic pigment structures with density of lichenoid infiltrate on histopathology

Density of lichenoid infiltrate	Chinese letter pattern	Diffuse	Reticular	$\chi 2^1$	P Value
None	3 (9.70)	3 (27.30)	3 (21.40)	5.653	0.463
Mild	22 (71.00)	5 (45.50)	8 (57.10)		
Moderate	3 (9.70)	3 (27.30)	2 (14.30)		
Severe	3 (9.70)	0 (0.00)	1 (7.10)		
Total	31 (100)	11(100)	14 (100)		

1Fisher's Exact test statistic

The data presents the association of Association of dermatoscopic pigment structures with density of lichenoid infiltrate on histopathology. The data indicates that the Mild density is the most frequently observed category across all patterns, particularly in the Chinese letter pattern (71%). The None category is most prevalent in the Diffuse pattern (27.3%), while the Severe density is rare across all patterns, with no cases (0) observed in the Diffuse pattern. There was no statistically significant ($\chi 2 = 5.653$, P= 0.463) difference between dermatoscopic pigment structures with density of lichenoid infiltrate on histopathology

Figure 22: Bar graph showing distribution of dermatoscopic pigment structures with density of lichenoid infiltrate on histopathology

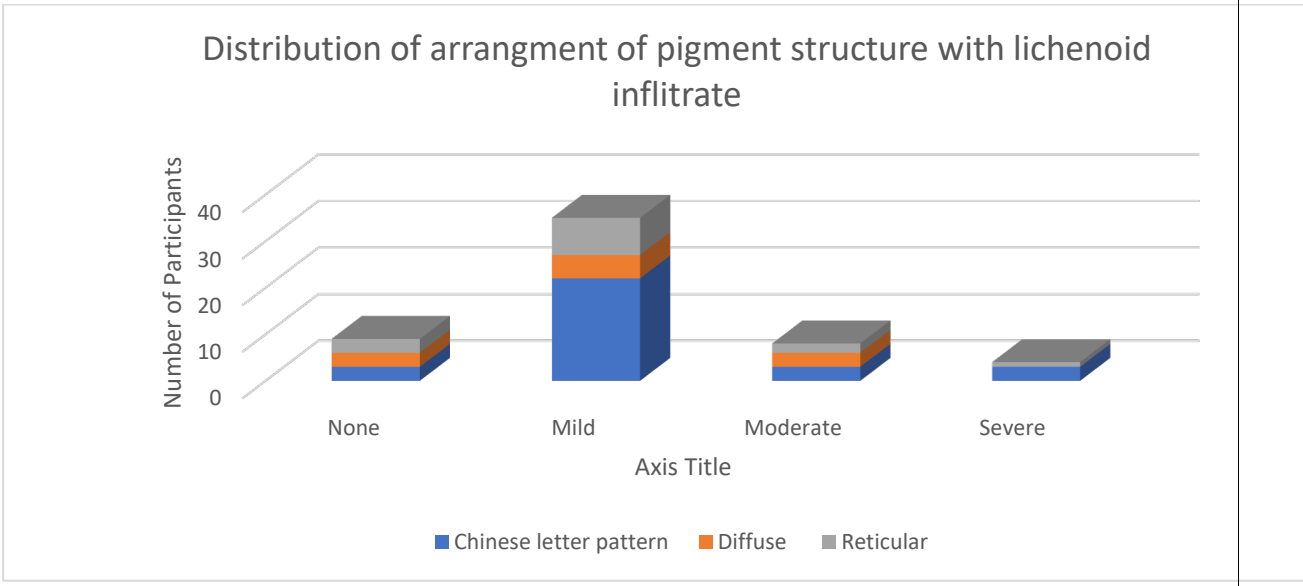


Table 23: "Association of Exaggeration of the normal pseudo reticular pigmentary network with Epidermal melanization"

Epidermal melanization	Exaggeration of the normal pseudo reticular pigmentary network Present	Exaggeration of the normal pseudo reticular pigmentary network Absent	$\chi 2^1$	P Value
Basal Layer only	7 (19.44)	2 (10.00)	8.264	0.016
1-3 layers	26 (72.22)	10 (50.00)		
>3 layers	3 (8.34)	8 (40.00)		
Total	36 (100)	20 (100)		

The association between the exaggeration of the normal pseudo-reticular pigmentary network and epidermal melanization was analyzed. Among the 36 patients with an exaggerated pseudo-reticular pigmentary network, the majority (72.22%) exhibited melanization extending to 1–3 layers of the epidermis. Melanization limited to the basal layer was observed in 7 patients (19.44%), while melanization involving more than 3 layers was seen in only 3 patients (8.34%). In comparison, greater proportion of the patients without an exaggerated pseudo-reticular pigmentary network, melanization was observed in more than 3 layers (40%). Melanization restricted to the basal layer was seen in only 2 patients (10%). A statistically significant association was found between epidermal melanization and the presence of an exaggerated pseudo-reticular pigmentary network ($\chi^2 = 8.264$, $P = 0.016$).

Figure 23: Bar graph showing distribution of epidermal melanization.

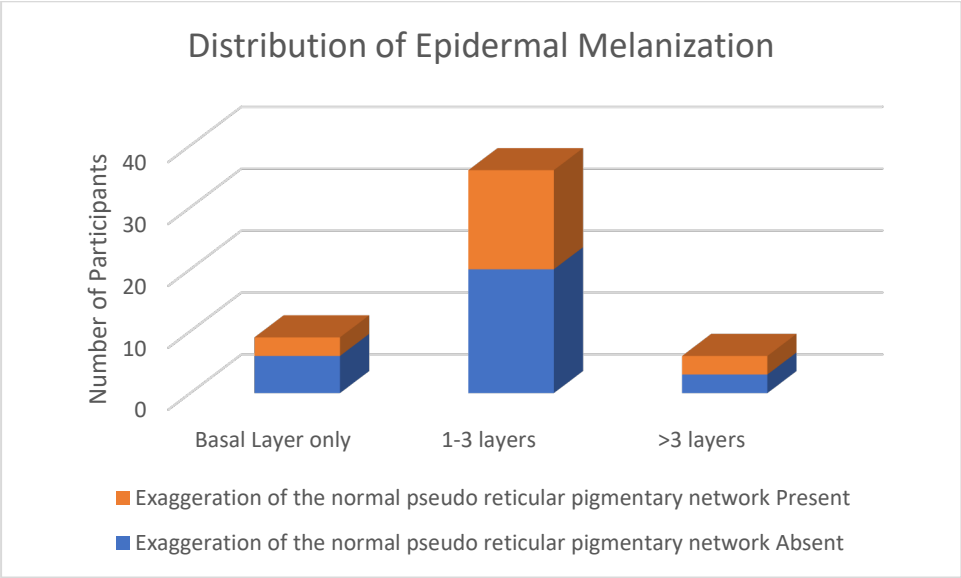


Table 24: "Association of Exaggeration of the normal pseudo reticular pigmentary network with Epidermal thickness"

Epidermal thickness	"Exaggeration of the normal pseudo reticular pigmentary network Present"	"Exaggeration of the normal pseudo reticular pigmentary network Absent"	$\chi 2^1$	P Value
Normal	6 (16.70)	8 (40.00)	4.050	0.132
Acanthosis	17 (47.20)	8 (40.00)		
Atrophy	13 (36.10)	4 (20.00)		
Total	36 (100)	20 (100)		

The association between the exaggeration of the normal pseudo-reticular pigmentary network and epidermal thickness was analyzed. Among the 36 patients with an exaggerated pseudo-reticular pigmentary network, the most common epidermal change observed was acanthosis, present in 17 patients (47.2%). Atrophy was noted in 13 patients (36.1%), while 6 patients (16.7%) had normal epidermal thickness. Among the 20 patients without an exaggerated pseudo-reticular pigmentary network, normal epidermal thickness and acanthosis were each observed in 8 patients (40%), while atrophy was present in 4 patients (20%). The association between the exaggeration of the pseudo-reticular pigmentary network and epidermal thickness was not statistically significant ($\chi^2 = 4.050$, $P = 0.132$).

Figure 24: Bar graph showing distribution of epidermal thickness.

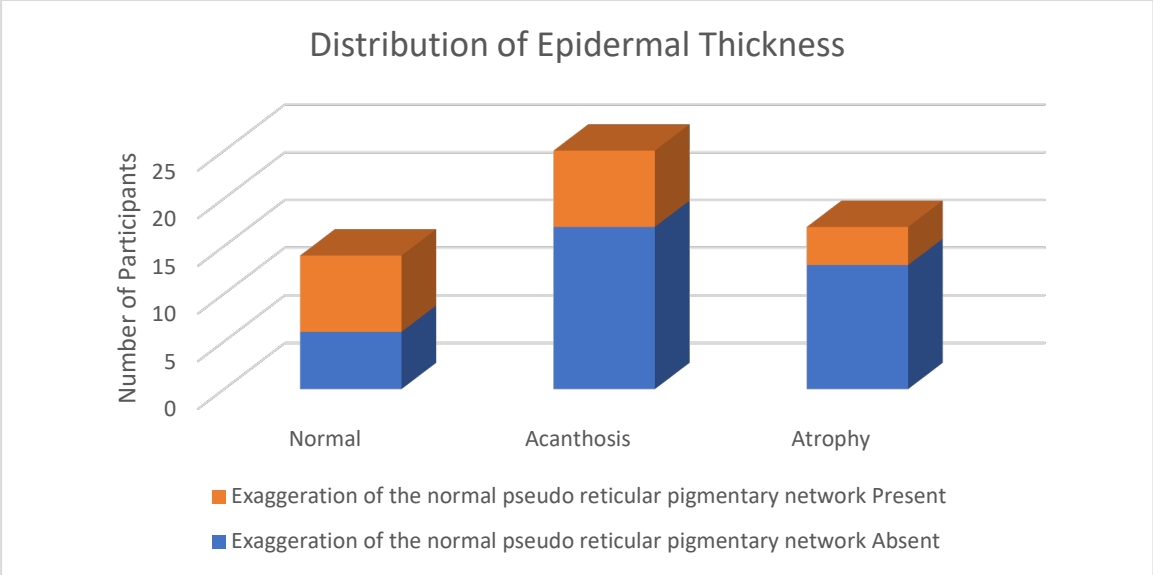


Table 25: Correlation of duration of disease with density of melanophages (per high power field 400×)

Parameter	r value (correlation coefficient)	P value
Correlation between duration of disease with density of melanophages	**0.862**	**0.0001**

The correlation between the duration of disease and the density of melanophages was found to be significant. r (54) = .862, p = 0.0001.

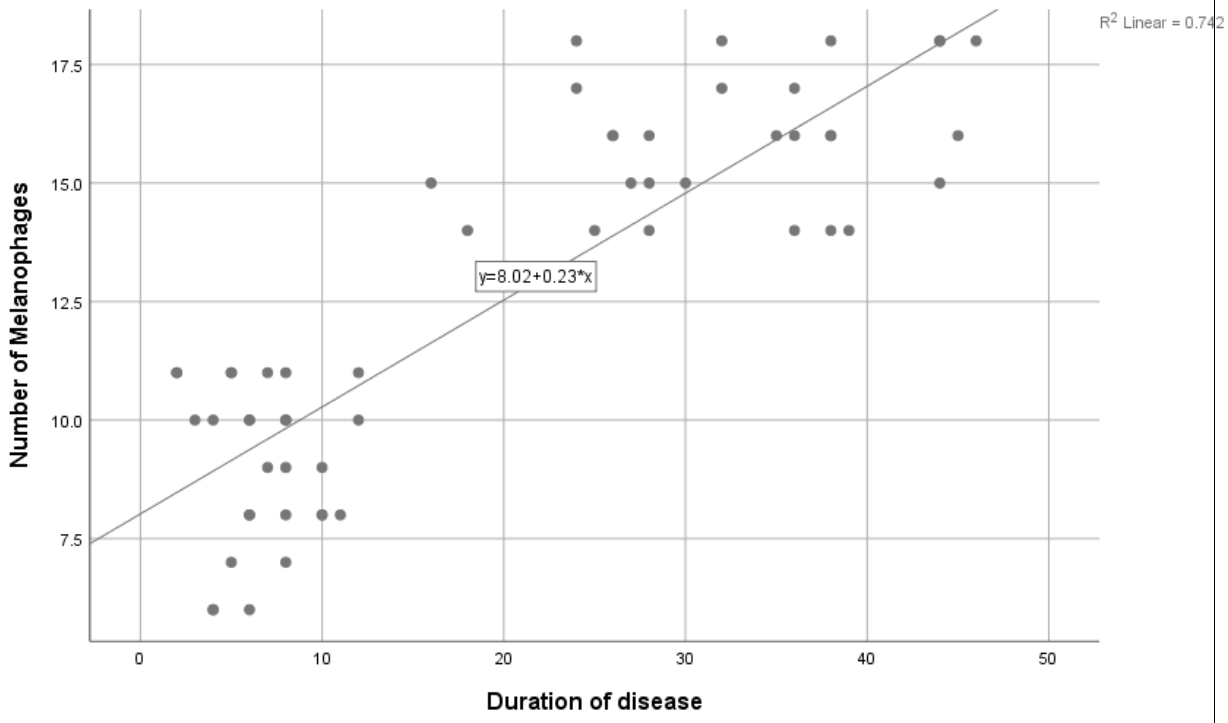


Figure 25:"Scatter plot depicting the relation between duration of disease and the density of melanophages in ADMH patients (n = 56). We can clearly see the relation between the duration of disease with density of melanophages is positive one, indicating that as the duration of disease increases, the density of melanophages will increase, and this increase can be attributed to 74% (R2 = 0.74) of the patients of ADMH in the study"

Discussion

Facial melanosis encompasses a variety of distinct conditions, all characterized by a common clinical manifestation of altered facial pigmentation. The resulting cosmetic disfigurement is readily apparent and can lead to considerable psychosocial impacts. The rise in patient awareness, coupled with the increased utilization of cosmetics and over-the-counter medications, has led to a significant and rapid growth in their incidence and relevance. This condition frequently manifests in patients from India, presenting intricate diagnostic and treatment challenges that involve clearly defined clinical entities.

Conditions that include ADMH, such as LPP, AD and RM, exhibit considerable overlapping characteristics, do not possess clear diagnostic guidelines, and are frequently challenging to distinguish both clinically and histopathologically. [6] There is a lack of global agreement regarding its naming conventions, and we believe that these disorders share a common pathological process as a collective group. Each of these cases illustrates a lichenoid reaction pattern resulting from a persistent subclinical injury of unclear origin. Other investigations have expressed comparable perspectives. [17,93]

Due to the presence of overlapping characteristics in numerous patients that do not exhibit clear clinical or histological distinctions, we opted not to further characterize these individuals, instead categorizing them under the broader term of ADMH. While melasma, amyloidosis, and post-inflammatory hyperpigmentation also manifest as macular hyperpigmentation, they can be distinguished from ADMH by the absence of interface dermatitis and/or the presence of previous inflammatory lesions.

The present study was carried out to evaluate the dermatoscopic characteristics of ADMH and to establish a correlation with histopathological findings.

The current investigation encompasses individuals aged 18 to 70 years, with the predominant age group being 31–40 years, representing nearly 40% of the subjects involved. In the investigation conducted by Solanki V et al.[4] the predominant age group impacted was 21–40 years, accounting for 53% of cases, which aligns with findings from present study. A study conducted by Agarwal P et al.[94] revealed that the age of patients ranged from 19 to 65 years, with a mean age of 35.3 years. This finding is comparable to the research by Hassan et al.[95] who reported a mean age of 27.40 years.

Among the study population in the present study, there were 34 female patients, representing 60.71% of the group, highlighting a notable female predominance in this analysis. In the investigation conducted by Solanki V et al.[4] female patients represented 62 cases (62%), a finding that aligns with the current study. The study conducted by Agarwal P et al.[94] also indicated that females were more affected than males, aligning with the findings of the current investigation. This observation may be attributed to hormonal factors, as well as the tendency for female patients to be more aware of their appearance compared to their male counterparts. The male predominance for ADMH observed in the research conducted by Sasidharanpillai S et al.[96] was inconsistent with the results of the current investigation.

Within the entire study population, 35 individuals were employed, representing 62.50% of the total population under investigation, which suggests that the majority of participants were engaged in employment in this analysis. In a comparable investigation conducted by Hassan et al.[95] housewives represented the largest segment (22.59%).

The findings of the present study reveal a distinct trend in the distribution of pigmentation, with the face emerging as the most commonly and universally affected area, succeeded by the upper limbs. The investigation conducted by Sasidharanpillai S et al. [96] revealed that ADMH was present on the face (21 patients, 70%), upper limbs (18 patients, 60%), upper trunk (10 patients, 33.3%), lower limbs (8 patients, 26.7%), neck (7 patients, 23.3%), and abdomen (3 patients, 10%).

Within the study population, the predominant pattern identified was pigmentation located on the forehead, malar, and retroauricular areas. The investigation conducted by Agarwal P et al. [94] identified centrofacial distribution as the predominant pattern in their study, aligning with the research conducted by Goh et al. [97] and Sanchez et al. [98]

The most prevalent type of pigmentation observed is greyish-black. This sombre tone may indicate a persistent condition, increased melanin accumulation in the skin, or external influences such as extended sun exposure or reactions to medications. The pigmentation observed in the investigation conducted by Sasidharanpillai S et al. [96] ranged from violaceous (eight, 26.7%) to grayish black/gray (20 patients, 66.7%), and brown (two patients, 6.7%).

Lichen Planus Pigmentosus (LPP) has been identified as the most prevalent cause, followed by Riehl's Melanosis and Ashy Dermatosis. In a study conducted by Solanki V et al.[4] melasma emerged as the predominant cause of facial melanosis, accounting for 49% of the total cases observed. A study conducted by Sobhanakumari K et al.[99] identified 8 distinct types of acquired melanosis within the study group. The conditions included melasma, Riehl's melanosis, AN, LPP, EDP, PIH, exogenous ochronosis, and Addison's disease. In their study, melasma emerged as the predominant form of acquired facial hypermelanosis, accounting for 63% of the cases observed. According

to the study conducted by Solanki V et al.[4] melasma was identified as the predominant cause of facial melanosis, accounting for 49% of the cases.

Dermoscopy is a non-invasive method that integrates digital photography with light microscopy to facilitate in vivo observation and diagnosis of pigmented skin lesions.

During dermatoscopic examination, we observed:

(i) the presence of dots, globules, and blotches in black, brown, and violet hues as the main pigment structures, which were further organized into dotted, Chinese letter, reticulate, or diffuse patterns;

(ii) the occurrence of telangiectasia;

(iii) a pronounced normal pseudoreticularpigmentary network;

(iv) structures resembling owl's eyes. Using these, we were able to further categorize the group into four levels of escalating disease severity, based on the examination of global dermatoscopic patterns and the related histopathological findings.

Similar to ours, the main dermatoscopic features observed in a study by Vinay et al.[5] included brownish-black and bluish-grey dots (n =42, 82.4%), globules (n =34, 66.7%), blotches (n =29, 56.9%), and telangiectasia (n =42, 82.4%). In this study, the predominant arrangement of pigment structures observed was the Chinese letter pattern, which aligns with the findings of Vinay et al.[5] The majority of patients exhibited an exaggeration of the normal pseudoreticularpigmentary network in the present study. Similarly in a study by Solanki V et al.[4] found that accentuated pseudopigment network was the most common dermoscopic pattern seen in their study. The second most prevalent observation was the presence of brown dots and globules in their study. Dermoscopy revealed a pseudo pigment network, dark brown to slate grey

dots and globules, telangiectasia, a worm-like pattern, and pigment obliterating follicular openings.

Various investigations have reported distinct histological observations in ADMH. The histology patterns are believed to differ according to the duration of the disease, and many of the entities categorized under ADMH represent various stages of the same condition. [15,73]

The histopathological characteristics of epidermal melanization revealed that a significant portion of the participants exhibited melanization in 1–3 layers (70.6%), aligning with the findings of Vinay et al. [5] Acanthosis was observed in the majority of cases (44.64%) regarding epidermal thickness in the current investigation. While observing the Upper dermal lichenoid infiltrate Mild (65.20%) cases were predominant which is similar to the study carried out by Vinay et al. [5]

No correlation ($\chi^2 = 4.050$, $P = 0.132$) was found between the dermatoscopic pigment structures and the depth of melanophages or the density of lichenoid infiltrate in the histopathological analysis, consistent with the findings of Vinay et al.[5] a notable correlation ($\chi^2 = 8.264$, $P = 0.016$) was identified between epidermal melanization and the existence of an enhanced pseudo-reticular pigmentary network. In a study conducted by Sharma VK et al.[100] the most prevalent dermoscopic observations were dots and/or globules, which showed a strong correlation with notable pigment incontinence observed in histological analysis.

The overall duration of the disease showed a positive correlation with the density of dermal melanophages and various levels of disease severity which is similar to the study carried out by Vinay et al.[5]

The findings suggest that the conditions in ADMH indicate an ongoing pathological process that evolves through different stages of lichenoid tissue damage, ultimately leading to progressive dermal pigment incontinence and the accumulation of melanophages over time. The initial phase is marked by localized vacuolization of the basal layer and slight pigment incontinence, which is observed under dermatoscopy as pigment dots alone. In the context of advancing disease and significant pigment incontinence, the dermis develops larger clusters of dermal melanophages, which manifest as globules and blotches during dermatoscopic examination. The density of dots and globules increases, leading to the formation of linear and semi-arcuate patterns. Initially, these patterns appear incomplete and fragmented, resembling Chinese characters, but they ultimately evolve into a complete reticulate pattern. In individuals experiencing ongoing damage, numerous dermal melanophages extensively infiltrate the dermis, disrupting the typical pigment structure. It is noteworthy that even in advanced stages, there is a preservation of eccrine and follicular openings. This observation warrants further investigation to understand the reasons behind the sparing of these structures. Among our cases, only one, which was clinically diagnosed as follicular LPP, exhibited involvement of hair follicular openings.

Conclusion

This investigation illustrates an uncomplicated approach to predict outcomes and advise individuals affected by a condition characterized by an extended clinical trajectory without a definitive cure. The relationship between clinical and dermatoscopic severity, along with the importance of serial dermatoscopy in tracking treatment response and disease progression, warrants additional investigation. The exploration of various conditions that may induce dermal melanosis, particularly those presenting as ADMH, is essential, alongside the investigation of disease processes that primarily lead to epidermal melanosis. Future investigations that include clinical, histological, and dermoscopic assessments could assist in clarifying the diagnosis and prognosis of conditions referred to as ADMH.

References

1. Taylor SC, Cook-Bolden F, Rahman Z, Strachan D. Acne vulgaris in skin of color. J AmAcad Dermatol. 2002;46(2 Suppl):S98–106.
2. Jablonski NG, Chaplin G. The evolution of human skin coloration. J Hum Evol.2000;39:57–106.
3. Shenoi SD, Prabhu S. Role of cultural factors in the biopsychosocial model of psychosomatic skin diseases: An Indian perspective. Clin Dermatol 2013;31:62-5.
4. Solanki V, Dongre A, Nayak C. A clinicoepidemiological study of different dermoscopic patterns in hyperpigmented facial lesions in a tertiary care centre. J Cutan Aesthet Surg 2024;17:112-23.
5. Vinay K, Bishnoi A, Parsad D, Saikia UN, Sendhil Kumaran M. Dermoscopic evaluation and histopathological correlation of acquired dermal macular hyperpigmentation. Int J Dermatol 2017;56:1395–9.
6. Chandran V, Kumarasinghe SP. Macular pigmentation of uncertain aetiology revisited: Two case reports and a proposed algorithm for clinical classification. Australas J Dermatol 2017;58:45–9.
7. Ebihara T, Nakayama H. Pigmented contact dermatitis. Clin Dermatol 1997;15:593–9.
8. Robles-Méndez JC, Rizo-Frías P, Herz-Ruelas ME, Pandya AG, Ocampo Candiani J. Lichen planus pigmentosus and its variants: Review and update. Int J Dermatol 2018;57:505–14.
9. Zaynoun S, Rubeiz N, Kibbi A-G. Ashy dermatoses – A critical review of the literature and a proposed simplified clinical classification. Int J Dermatol 2008;47:542–4.

10. Wang L, Xu AE. Four views of Riehl's melanosis: clinical appearance, dermoscopy, confocal microscopy and histopathology. J Eur Acad Dermatol Venereol 2014; 28:1199–1206.
11. Pirmez R, Duque-Estrada B, Donati A, et al. Clinical and dermoscopic features of lichen planus pigmentosus in 37 patients with frontal fibrosing alopecia. Br J Dermatol 2016; 175: 1387–1390.
12. Ramirez CO. Los cenicientos problema clinico. In: Proceedings of the First Central American Congress of Dermatology, San Salvador, 5–8 December, 1957. San Salvador: Central American Dermatological Society, 1957: 122–130.
13. Convit J, Kerdel-Vegas F, Rodriguez G. Erythema dyschromicum perstans: a hitherto undescribed skin disease. J Invest Dermatol 1961; 36: 459–462.
14. Bhutani LK, Bedi TR, Pandhi RK, et al. Lichen planus pigmentosus. Dermatologica 1974; 149: 43–45.
15. Vega ME, Waxtein L, Arenas R, et al. Ashy dermatosis and lichen planus pigmentosus: a clinicopathologic study of 31 cases. Int J Dermatol 1992; 31: 90–94.
16. Naidorf KF, Cohen SR. Erythema dyschromicum perstans and lichen planus. Arch Dermatol 1982; 118: 683–685.
17. Bhutani LK. Ashy dermatosis or lichen planus pigmentosus: what is in a name? Arch Dermatol 1986; 122: 133.
18. Zaynoun S, Rubeiz N, Kibbi AG. Ashy dermatoses – a critical review of the literature and a proposed simplified clinical classification. Int J Dermatol 2008; 47: 542–544.

19. Chandran V, Kumarasinghe SP. Macular pigmentation of uncertain aetiology revisited: two case reports and a proposed algorithm for clinical classification. Australas J Dermatol 2017;58: 45–49.
20. Anderson A, Wood B, Kumarasinghe SP. Defining erythema dyschromicum perstans, ashy dermatosis, lichen planus pigmentosus and idiopathic eruptive macular pigmentation: a global consensus needed. Sri Lanka J Dermatol 2012; 16: 20–22.
21. Sanz de Galdeano C, Leaute-Labreze C, Bioulac-Sage P, et al. Idiopathic eruptive macular pigmentation: report of 5 patients. Pediatr Dermatol 1996; 13: 274–277.
22. Joshi R. Idiopathic eruptive macular pigmentation with papillomatosis: report of nine cases. Indian J Dermatol Venereol 2007; 73: 402–405.
23. Sandhu S, Neema S, Radhakrishnan S. Dermoscopy of disorders of hyperpigmentation. Pigment Int 2021;8:14-24.
24. Wang L, Xu AE. Four views of Riehl's melanosis: clinical appearance, dermoscopy, confocal microscopy and histopathology. J Eur Acad Dermatol Venereol 2014; 28: 1199–1206.
25. Sandhu S, Neema S, Radhakrishnan S. Dermoscopy of disorders of hyperpigmentation. Pigment Int 2021;8:14-24.
26. Verma SB. Redefining color of Indian skin. J Eur Acad Dermatol Venereol 2008;22:1263-4.
27. Jaswal IJ. Pigmentary variation in Indian populations. Acta Anthropogenet 1983;7:75-83.
28. Jaswal IJ. Skin color in north indian populations. J Hum Evol 1979;8:361-6.

29. Verma SB. Dermatology for the elderly: An Indian perspective. Clin Dermatol 2011;29:91-6.
30. Reich D, Thangaraj K, Patterson N, Price AL, Singh L. Reconstructing Indian population history. Nature 2009;461:489-94.
31. Cayce AK, Feldman RS, McMichael AJ. Hyperpigmentation: a review of common treatment options. J Drugs Dermatol 2004;3(6):668-673.
32. Fatima S, Braunberger T, Mohammad T, Kohli I, Hamzavi I. The role of sunscreen in melasma and postinflammatory hyperpigmentation. Indian J Dermatol. 2020;65(1):5-10.
33. Cestari T, Dantas L, Boza J. Acquired hyperpigmentations. An Bras Dermatol. 2014;89(1):11-25.
34. Nouveau S, Agrawal D, Kohli M, Bernerd F, Misra N, Nayak C. Skin hyperpigmentation in Indian population: Insights and best practice. Indian J Dermatol. 2016;61(5):487-95.
35. Simon JD, Peles D, Wakamatsu K, Ito S. Current challenges in understanding melanogenesis: Bridging chemistry, biological control, morphology, and function. Pigment Cell Melanoma Res 2009;22:563-79.
36. Meredith P, Riesz J. Radiative relaxation quantum yields for synthetic eumelanin. Photochem Photobiol 2004;79:211-6.
37. Del Bino S, Bernerd F. Variations in skin colour and the biological consequences of ultraviolet radiation exposure. Br J Dermatol 2013;169 Suppl 3:33-40.
38. Jablonski NG, Chaplin G. Colloquium paper: Human skin pigmentation as an adaptation to UV radiation. Proc Natl Acad Sci U S A 2010;107 Suppl 2:8962-8.

39. Hourblin V, Nouveau S, Roy N, de Lacharrière O. Skin complexion and pigmentary disorders in facial skin of 1204 women in 4 Indian cities. Indian J Dermatol Venereol Leprol 2014;80:395-401.
40. Mukherjee M, Mukerjee S, Sarkar-Roy N, Ghosh T, Kalpana D, Sharma AK. Polymorphisms of four pigmentation genes (SLC45A2, SLC24A5, MC1R and TYRP1) among eleven endogamous populations of India. J Genet 2013;92:135-9.
41. Nicolaidou E, Katsambas AD. Pigmentation disorders: hyperpigmentation and hypopigmentation. Clin Dermatol. 2014 Jan-Feb;32(1):66-72.
42. Cutri FT, Ruocco E, Pettinato G, Ciancia G. Lichen planus pigmentosus-like ashy dermatosis. Dermatol Reports. 2011;3:e46.
43. Torrelo A, Zaballos P, Colmenero I, Mediero IG, de Prada I, Zambrano A. Erythema dyschromicum perstans in children: a report of 14 cases. J Eur Acad Dermatol Venereol. 2005;19:422–6.
44. Schwartz RA. Erythema dyschromicum perstans: the continuing enigma of Cinderella or ashy dermatosis. Int J Dermatol. 2004;43:230–2.
45. Osswald SS, Proffer LH, Sartori CR. Erythema dyschromicum perstans: a case report and review. Cutis. 2001;68:25–8.
46. Silverberg NB, Herz J, Wagner A, Paller AS. Erythema dyschromicum perstans in prepubertal children. Pediat Dermatol. 2003;20:398–403.
47. Ummap PS. Erythema dyschromicum perstans. Indian J Dermatol. 1996;41:68–9.
48. Keisham C, Sarkar R, Garg VK, Chugh S. Ashy dermatosis in an 8-year-old Indian child. Indian Dermatol Online J. 2013;4:30-2.

49. Numata T, Harada K, Tsuboi R, Mitsuhashi Y. Erythema Dyschromicum Perstans: Identical to Ashy Dermatosis or Not? Case Rep Dermatol. 2015;7:146-50.
50. Ghosh A, Coondoo A. Lichen Planus Pigmentosus: The Controversial Consensus. Indian J Dermatol. 2016;61:482-6.
51. Elmas OF, Acar EM, Kilitçi A. Dermoscopic diagnosis of ashy dermatosis: a retrospective study. Indian Dermatol Online J 2019;10:639-43.
52. Errichetti E, Angione V, Stinco G. Dermoscopy in assisting the recognition of ashy dermatosis. JAAD Case Rep 2017;3:482-4.
53. Gupta V, Sharma VK. Ashy dermatosis, lichen planus pigmentosus and pigmented cosmetic dermatitis: are we splitting the hair? Indian J Dermatol Venereol Leprol 2018;84:470-4.
54. Tlougan BE, Gonzalez ME, Mandal RV, Kundu RV, Skopicki DL. Erythema dyschromicum perstans. Dermatol Online J. 2010:16:17.
55. Mahajan VK, Chauhan PS, Mehta KS, Sharma AL. Erythema Dyschromicum Perstans: Response to Topical Tacrolimus. Indian J Dermatol. 2015;60:525.
56. Kroon MW, Wind BS, Meesters AA, Wolkerstorfer A, van der Veen JPW, Bos JD, et al. Non-ablative 1550 nm fractional laser therapy not effective for erythema dyschromicum perstans and postinflammatory hyperpigmentation: a pilot study. J Dermatolog Treat. 2012;23:339-344.
57. Piquero-Martin J, Pearez-Alfonzo R, Abrusci V, Briceno L, Gross A, Mosca W, Tapia F, Convit J. Clinical trial with clofazimine for treating erythema dyschromicum perstans: evaluation of cellmediated immunity. Int J Dermatol. 1989;28:198.

58. Baranda L, Torres-Alavarez B, Cortes-Franco R, Moncada B, Portales-Perez DP, Gonzalez-Amaro R. Involvement of cell adhesion and activation molecules in the pathogenesis of erythema dyschromicum perstans (ashy dermatitis): the effect of clofazimine therapy. Arch Dermatol. 1997;133:325.
59. Kontochristopoulos G, Stavropoulos P, Panteleos D, Aroni K. Erythema dyschromicum perstans: response to dapsone therapy. Int J Dermatol. 1998;37:796.
60. Bahadir S, Cobanoglu U, Cimsit G, Yayli S, Alpay K. Erythema Dyschromicum Perstans: Response to Dapsone therapy: Int J Dermatol. 2004;43:220–2.
61. Bhutani LK, George M, Bhate SM. Vitamin A in the treatment of lichen planus pigmentosus. British J Dermatol. 1979;100:473–5.
62. Rieder E, Kaplan J, Kamino H, Sanchez M, Pomeranz MK. Lichen planus pigmentosus. Dermatol Online J 2013;19:20713.
63. Sehgal VN, Verma P, Bhattacharya SN, Sharma S, Rasool F. Lichen planus pigmentosus. Skinmed 2013;11:96-103.
64. Pock L, Jelínková L, Drlík L, Abrhámová S, Vojtechovská S, Sezemská D, et al. Lichen planus pigmentosus-inversus. J Eur Acad Dermatol Venereol 2001;15:452-4.
65. Tienthavorn T, Tresukosol P, Sudtikoonaseth P. Patch testing and histopathology in Thai patients with hyperpigmentation due to erythema dyschromicum perstans, lichen planus pigmentosus, and pigmented contact dermatitis. Asian Pac J Allergy Immunol 2014;32:185-92.
66. Kanwar AJ, Dogra S, Handa S, Parsad D, Radotra BD. A study of 124 Indian patients with lichen planus pigmentosus. Clin Exp Dermatol 2003;28:481-5.

67. Al-Mutairi N, El-Khalawany M. Clinicopathological characteristics of lichen planus pigmentosus and its response to tacrolimus ointment: An open label, non-randomized, prospective study. J Eur Acad Dermatol Venereol 2010;24:535-40.
68. Vachiramon V, Suchonwanit P, Thadanipon K. Bilateral linear lichen planus pigmentosus associated with hepatitis C virus infection. Case Rep Dermatol 2010;2:169-172.
69. Hong S, Shin JH, Kang HY. Two cases of lichen planus pigmentosus presenting with a linear pattern. J Korean Med Sci 2004;19:152-4.
70. Kumar YH, Babu AR. Segmental lichen planus pigmentosus: An unusual presentation. Indian Dermatol Online J 2014;5:157-9.
71. Friedman P, Sabban EC, Marcucci C, Peralta R, Cabo H. Dermoscopic findings in different clinical variants of lichen planus. Is dermoscopy useful? Dermatol Pract Concept 2015;5:51-5.
72. Murzaku EC, Bronsnick T, Rao BK. Axillary lichen planus pigmentosus-inversus: Dermoscopic clues of a rare entity. Diagnosis: Lichen planus pigmentosus (LPP). J Am Acad Dermatol 2014;71:e119-20.
73. Kumarasinghe, S. P. W., Pandya, A., Chandran, V., Rodrigues, M., Dlova, N. C., Kang, H. Y., Ramam, M., Dayrit, J. F., Goh, B. K., & Parsad, D. (2019). A global consensus statement on ashy dermatosis, erythema dyschromicum perstans, lichen planus pigmentosus, idiopathic eruptive macular pigmentation, and Riehl's melanosis. International Journal of Dermatology, 58(3), 263–272.
74. Pérez-Bernal, A., Muñoz-Pérez, M. A., & Camacho, F. (2000). Management of Facial Hyperpigmentation. American Journal of Clinical Dermatology, 8, 261–268.

75. Rorsman, H. (1982). Riehl's melanosis. International Journal of Dermatology, 21(2), 75–78.
76. Minami, S., & Noma, Y. (1950). Melanosis faciei feminae. Dermatol Urol, 12, 73–77.
77. Pires MC, Manoel Silva dos Reis V, Mitelmann R, Moreira F. Pigmented contact dermatitis due to Plathymenia foliosa dust. Contact Dermatitis 1999;40:339.
78. Bishnoi A, Vinay K, Arshdeep, Parsad D, Handa S, Saikia UN, et al. Contact sensitization to hair colours in acquired dermal macular hyperpigmentation: Results from a patch and photo-patch test study of 108 patients. J Eur Acad Dermatology Venereol 2019;33:1349–57.
79. Khanna N, Rasool S. Facial melanoses: Indian perspective. Indian J Dermatology, Venereol Leprol 2011;77:552-63.
80. Sugai T, Takahasmi Y, Takagi T. Pigmented cosmetic dermatitis and coal tar dyes *. Contact Dermatitis 1977;3:249–56.
81. Honigman, A., & Rodrigues, M. (2022). Differential diagnosis of melasma and hyperpigmentation. Dermatological Reviews, der2.144, 30–37.
82. Yim, J. H., Kang, I.-H., Shin, M. K., & Lee, M.-H. (2019). Differences among Dermoscopic findings in Riehl's melanosis of the cheek and neck. Annals of Dermatology, 31(4), 460–463.
83. Krueger, L., Saizan, A., Stein, J. A., & Elbuluk, N. (2022). Dermoscopy of acquired pigmentary disorders: A comprehensive review. International Journal of Dermatology, 61(1), 7–19.
84. Park, J.-Y., Kim, Y. C., Lee, E.-S., Park, K.-C., & Kang, H. Y. (2012). Acquired bilateral melanosis of the neck in perimenopausal women. The British Journal of Dermatology, 166(3), 662–665.

85. Kim, S. M., Lee, E.-S., Sohn, S., & Kim, Y. C. (2020). Histopathological features of Riehl melanosis. The American Journal of Dermatopathology, 42(2), 117–121.
86. Chan, K. T. M., & Ma, M. M. W. (2019). A case of an elderly women presented with severe pigmented contact dermatitis (Riehl's melanosis) effectively treated with topical lignin peroxidase cream. Medical Reports & Case Studies, 4(1), 1–4.
87. Choi, M. E., Lim, Y., Lee, W. J., Won, C. H., Lee, M. W., & Chang, S. E. (2022). Characteristics of Riehl melanosis in an ethnic Asian population: A comparative study according to age, sex, and hair dye use. JEADV Clinical Practice, 1(3), 219–228.
88. Kim, K. M., & Lim, H. W. (2022). The uses of tranexamic acid in dermatology: A review. International Journal of Dermatology, 62, 589–598.
89. Shimshak, S. J. E., Tolaymat, L. M., Haga, C. B., Dawson, N. L., Gillis, M. S., Yin, M., Kirsch, B., Cooper, M., & Sluzevich, J. C. (2022). A review of Oral therapies for the treatment of skin hyperpigmentation. Journal of Cutaneous Medicine and Surgery, 26(2), 169–175.
90. Rani, S., & Ahuja, A. (2022). Chemical peel as an adjuvant treatment in pigmented contact dermatitis: A case series. Journal of Cosmetic and Laser Therapy, 24(6–8), 112–117.
91. Argenziano G, Soyer HP, Chimenti S, et al. Dermoscopy of pigmented skin lesions: results of a consensus meeting via the Internet. J Am Acad Dermatol 2003; 48: 679–693.
92. Campos-do-Carmo G, Ramos-e-Silva M. Dermoscopy: basic concepts. Int J Dermatol 2008; 47: 712–719.

93. Bhutani LK. Pigmented contact dermatitis vs lichen planus pigmentosus. Int J Dermatol 1977; 16: 860–862.
94. Agarwal P, Gajjar K, Mistry A. A clinico-epidemiological study of facial melanosis at a tertiary care center in Gujarat. Pigment Int 2019;6:96-101.
95. Hassan I, Aleem S, Bhat YJ, Anwar P. A clinico-epidemiological study of facial melanosis. Pigment Int 2015;2:34-40.
96. Sasidharanpillai S, Govindan A, Ajithkumar K, Mahadevan ST, Bindu V, Khader A, et al. Histological evaluation of acquired dermal macular hyperpigmentation. Indian Dermatol Online J 2019;10:542-6.
97. Varma K., et al. A role of estrogen in etiopathogenesis of melasma in female patients: A prospective observational study in a tertiary care hospital. Indian J Clin Exp Dermatol 2015;1:21-4.
98. Grimes PE. Melasma: etiologic and therapeutic considerations. Arch Dermatol 1995;131:1453-7.
99. Sobhanakumari K, Issac CM, Ananthavally A. Study of acquired facial hyperpigmentation. J. Evid. Based Med. Healthc. 2017; 4(34), 2026-2031.
100. Sharma VK, Gupta V, Pahadiya P, Vedi KK, Arava S, Ramam M. Dermoscopy and patch testing in patients with lichen planus pigmentosus on face: A cross-sectional observational study in fifty Indian patients. Indian J Dermatol Venereol Leprol 2017;83:656-62.

ANNEXURE I- INFORMED CONSENT FORM

"STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE"

Name of Student/Principal Investigator: DR. M. SAHITHI

Name of Guide/Co Investigators: DR. SHIVAKUMAR PATIL

Introduction: Hyperpigmentary skin disorders can be defined as the disorders that are caused due to increased pigmentation of the skin or mucous membrane.

Acquired dermal macular pigmentation (ADMH) is a broad term that includes disorders which are clinically characterized by small and large pigmented macules or patches and histopathologically show current or resolved interface dermatitis with pigment incontinence, without any clinically evident prior inflammatory skin lesions.

Explanation of procedure:
After screening, eligible study participants are chosen. Detailed history will be taken and clinical examination of the patient will be done and informaed consent will be taken. Digital photographs of the lesions on the face will be taken using identical camera settings, patients positioning and lighting at baseline. Dermoscopy will be done using a dermoscope, which is like a magnifying glass, on a single lesion on the face and pictures will be taken. Then a 4mm skin biopsy will be taken from the site where dermoscopy was done under aseptic precautions and seen under the microscope.

Withdrawal from participation in the study:

Participation in this study in voluntary.You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by

participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to preventany person to identify you. Your identity will never be revealed. The data collected from youwill be kept confidential and only processed or aggregated data will be used for publication. **Financial incentives:** You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator.**

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: **"M.Sahithi, mobile number: +91-7337456787, email ID:**

sahithimarineni13@gmail.com" If you have any question or complaints with regardto your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**STUDY OF ASSOCIATION OF DERMOSCOPIC FINDINGS AND HISTOPATHOLOGICAL CHANGES IN ACQUIRED DERMAL MACULAR HYPERPIGMENTED DISORDERS OF FACE**". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

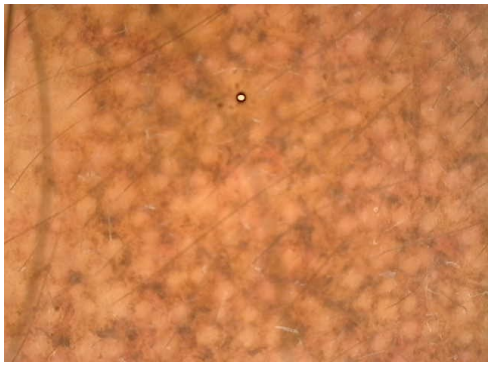
ANNEXURE II- PROFORMA

	Demographic details	
DATE		
NAME		
OP NUMBER		
IP NUMBER		
AGE		
SEX	**FEMALE**	**MALE**
ADDRESS WITH PHONE NUMBER		
MARITAL STATUS	**MARRIED**	**UNMARRIED**
OCCUPATION	**EMPLOYED**	**UNEMPLOYED**
CHIEF COMPLAINTS		
DISEASE DURATION	**YEARS**	**MONTHS**
H/O TREATMENT		
SYSTEMIC	**PRESENT**	**ABSENT**
MENTION (IF ANY)		

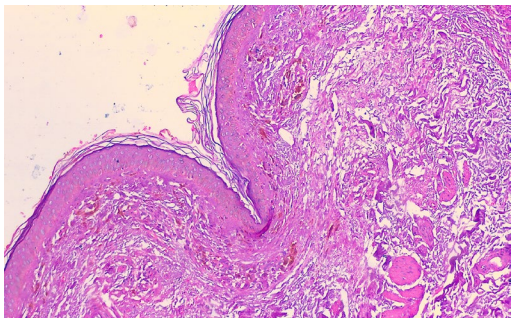
TOPICAL	**PRESENT**	**ABSENT**
MENTION (IF ANY)		
FAMILY HISTORY	**PRESENT**	**ABSENT**
GENERAL EXAMINATION		
PR	**bpm**	
BP	**mm/hg**	
WEIGHT	**Kg**	
HEIGHT	**cm**	
BMI		
SITE OF LESIONS		
DIAGNOSIS		

ANNEXURE III- PHOTOGRAPHS

CASE-1

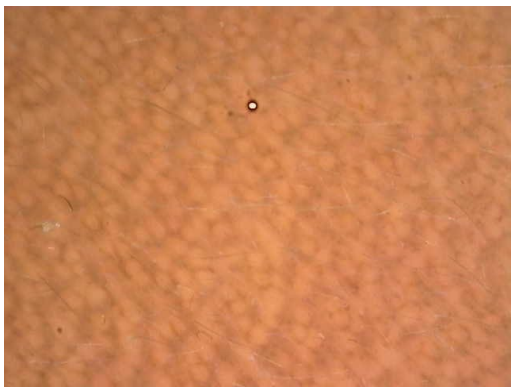


The image shows brown dots, reticulate pattern of pigmentation.

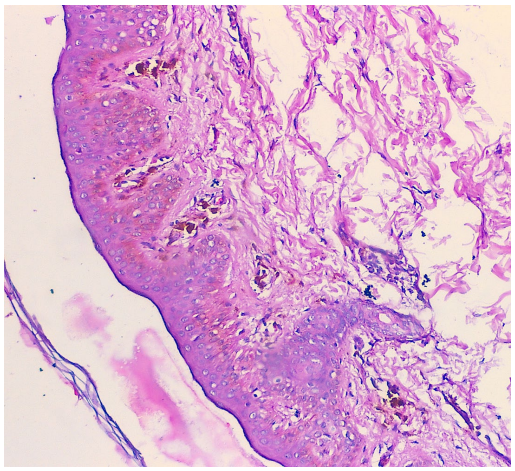


The HPE image shows flattening of rete redges, inflammatory infiltrate in the dermis.

CASE-2



Reticulate pattern of pigmentation is seen in the dermoscopy with dots and globules present.



In the HPE, rete ridges appear to be normal, with pigment incontinence in the dermis and inflammatory infiltrate present.

S.No.	Age	Sex	Marital Status	Comorbidity	Type of Residence	Diagnosis	Employment status	Location of Pigmentation				
1	24	Female	Married	DM	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	Upper trunk	Lower limbs	Neck
2	34	Male	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
3	35	Female	Married	HTN	Urban	Riehls melanosis	Unemployed	Face	Upper limb	N/A	Lower limbs	N/A
4	26	Female	Married	Absent	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	N/A	N/A	N/A
5	43	Male	Married	DM	Urban	Ashy dermatitis	Employed	Face	Upper limb	N/A	N/A	N/A
6	37	Male	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	Lower limbs	N/A
7	45	Female	Married	DM	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	Upper trunk	N/A	N/A
8	25	Male	Married	Absent	Urban	Riehls melanosis	Unemployed	Face	Upper limb	N/A	N/A	Neck
9	47	Female	Married	DM	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	Upper trunk	N/A	N/A
10	33	Male	Married	Absent	Urban	Ashy dermatitis	Employed	Face	N/A	Upper trunk	N/A	N/A
11	46	Female	Married	HTN	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
12	27	Male	Unmarried	Absent	Urban	Lichen planus pigmentosus	Employed	Face	N/A	Upper trunk	N/A	Neck
13	48	Female	Married	Absent	Urban	Lichen planus pigmentosus	Unemployed	Face	N/A	Upper trunk	N/A	N/A
14	31	Male	Married	Absent	Rural	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	Lower limbs	Neck
15	26	Female	Unmarried	DM	Urban	Ashy dermatitis	Employed	Face	N/A	Upper trunk	N/A	N/A
16	48	Male	Married	Absent	Rural	Lichen planus pigmentosus	Employed	Face	N/A	Upper trunk	N/A	N/A
17	35	Female	Unmarried	Hypothyroidism	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	N/A	N/A	N/A
18	55	Male	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	Lower limbs	Neck
19	38	Female	Unmarried	HTN	Rural	Ashy dermatitis	Employed	Face	Upper limb	N/A	N/A	N/A
20	28	Female	Married	Absent	Urban	Riehls melanosis	Employed	Face	Upper limb	N/A	N/A	N/A
21	44	Female	Married	DM	Rural	Riehls melanosis	Employed	Face	N/A	Upper trunk	N/A	N/A
22	33	Female	Unmarried	Absent	Urban	Riehls melanosis	Unemployed	Face	Upper limb	Upper trunk	N/A	N/A
23	49	Female	Married	Hypothyroidism	Rural	Lichen planus pigmentosus	Unemployed	Face	N/A	N/A	N/A	Neck
24	39	Male	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	N/A	N/A	N/A	N/A
25	48	Female	Unmarried	Hypothyroidism	Rural	Ashy dermatitis	Employed	Face	N/A	Upper trunk	N/A	Neck
26	55	Male	Married	Absent	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	N/A	Lower limbs	N/A
27	26	Male	Married	HTN	Urban	Riehls melanosis	Employed	Face	N/A	Upper trunk	N/A	N/A
28	35	Female	Unmarried	Absent	Urban	Riehls melanosis	Employed	Face	Upper limb	N/A	Lower limbs	N/A
29	45	Male	Married	Absent	Urban	Riehls melanosis	Employed	Face	N/A	Upper trunk	N/A	N/A
30	22	Female	Unmarried	DM	Rural	Ashy dermatitis	Unemployed	Face	N/A	Upper trunk	N/A	Neck
31	35	Male	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
32	36	Female	Married	Hypothyroidism	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
33	34	Male	Unmarried	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	Lower limbs	N/A
34	52	Female	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
35	28	Male	Unmarried	Absent	Rural	Ashy dermatitis	Employed	Face	Upper limb	N/A	Lower limbs	N/A
36	53	Female	Married	HTN	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	N/A	N/A	N/A
37	33	Female	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
38	57	Male	Married	Absent	Rural	Lichen planus pigmentosus	Employed	Face	N/A	Upper trunk	N/A	Neck
39	34	Female	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
40	28	Female	Unmarried	DM	Rural	Ashy dermatitis	Employed	Face	Upper limb	N/A	N/A	N/A
41	58	Female	Married	Absent	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	N/A	N/A	N/A
42	36	Male	Married	Absent	Urban	Riehls melanosis	Employed	Face	Upper limb	N/A	N/A	Neck
43	33	Female	Married	HTN	Rural	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A

44	59	Male	Married	Absent	Urban	Ashy dermatitis	Unemployed	Face	Upper limb	N/A	N/A	N/A
45	27	Female	Unmarried	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
46	63	Female	Married	Hypothyroidism	Rural	Riehls melanosis	Unemployed	Face	Upper limb	N/A	N/A	N/A
47	35	Male	Unmarried	Absent	Urban	Riehls melanosis	Employed	Face	Upper limb	N/A	N/A	Neck
48	68	Male	Married	Absent	Urban	Lichen planus pigmentosus	Unemployed	Face	Upper limb	N/A	N/A	N/A
49	22	Female	Unmarried	DM	Urban	Ashy dermatitis	Employed	Face	Upper limb	N/A	N/A	N/A
50	54	Female	Married	Absent	Urban	Ashy dermatitis	Unemployed	Face	N/A	Upper trunk	N/A	N/A
51	37	Male	Married	Absent	Urban	Ashy dermatitis	Employed	Face	Upper limb	N/A	N/A	N/A
52	56	Female	Married	HTN	Urban	Lichen planus pigmentosus	Employed	Face	N/A	N/A	N/A	N/A
53	38	Female	Married	Absent	Urban	Lichen planus pigmentosus	Employed	Face	Upper limb	N/A	N/A	N/A
54	32	Female	Unmarried	Absent	Rural	Lichen planus pigmentosus	Unemployed	Face	N/A	N/A	N/A	Neck
55	32	Female	Unmarried	DM	Rural	Riehls melanosis	Unemployed	Face	Upper limb	N/A	N/A	N/A
56	68	Female	Married	Absent	Urban	Riehls melanosis	Unemployed	Face	Upper limb	N/A	N/A	N/A

Distribution of Pigmentation	Color of Pigmentation	Dermatoscopic feature							Arrangement of Pigment structure
forehead, retroauricular	Brown	Dots	Globules	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Centrofacial	Grayish/Black	Absent	Absent	Absent	Telangiectasia	Eccrine opening	Absent	Peri-follicular	Reticular
Centrofacial	Grayish/Black	Absent	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Violaceous	Dots	Absent	Difffuse	Absent	Eccrine opening	owl	Peri-follicular	Reticular
forehead, retroauricular	Grayish/Black	Dots	Globules	Difffuse	Telangiectasia	Eccrine opening	Absent	Absent	Reticular
forehead, retroauricular	Violaceous	Absent	Absent	Absent	Absent	Eccrine opening	owl	Peri-follicular	Reticular
forehead, retroauricular	Grayish/Black	Dots	Globules	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Absent	Telangiectasia	Eccrine opening	Absent	Peri-follicular	Reticular
Forehead,malar, retroauricular	Grayish/Black	Absent	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Diffuse
Forehead,malar, retroauricular	Grayish/Black	Absent	Absent	Absent	Absent	Eccrine opening	owl	Absent	Chinese letter arrangement
Forehead,malar, retroauricular	Violaceous	Dots	Globules	Difffuse	Absent	Eccrine opening	owl	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Absent	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Globules	Absent	Telangiectasia	Eccrine opening	Absent	Peri-follicular	Diffuse
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Absent	Chinese letter arrangement
Forehead,malar, retroauricular	Brown	Absent	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Diffuse
Forehead,malar, retroauricular	Brown	Dots	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Absent	Absent	Absent	Telangiectasia	Eccrine opening	Absent	Absent	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Globules	Absent	Absent	Absent	owl	Peri-follicular	Reticular
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Grayish/Black	Absent	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Diffuse
forehead, retroauricular	Grayish/Black	Dots	Globules	Absent	Telangiectasia	Absent	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Violaceous	Dots	absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Grayish/Black	Dots	absent	Absent	Absent	Eccrine opening	Absent	Absent	Diffuse
forehead, retroauricular	Grayish/Black	Absent	absent	Absent	Absent	Eccrine opening	owl	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Grayish/Black	Dots	Globules	Absent	Telangiectasia	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Grayish/Black	Absent	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Grayish/Black	Absent	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead, retroauricular	Grayish/Black	Dots	Absent	Absent	Absent	Absent	Absent	Absent	Chinese letter arrangement
Perioral	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Reticular
Centrofacial	Grayish/Black	Absent	Absent	Absent	Telangiectasia	Eccrine opening	Absent	Peri-follicular	Diffuse
Periorbital	Violaceous	Dots	Globules	Absent	Absent	Eccrine opening	owl	Peri-follicular	Reticular
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Malar, retroauricular	Grayish/Black	Absent	Absent	Absent	Absent	Eccrine opening	Absent	Absent	Diffuse
Malar, retroauricular	Violaceous	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Malar, retroauricular	Grayish/Black	Dots	Globules	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Reticular
Malar, retroauricular	Brown	Dots	Absent	Absent	Telangiectasia	Eccrine opening	owl	Peri-follicular	Chinese letter arrangement
Malar, retroauricular	Grayish/Black	Absent	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Periorbital	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Absent	Chinese letter arrangement
Centrofacial	Grayish/Black	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Diffuse
Centrofacial	Grayish/Black	Absent	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Malar	Brown	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Reticular

forehead	Violaceous	Absent	Absent	Absent	Telangiectasia	Eccrine opening	Absent	Absent	Diffuse
forehead	Grayish/Black	Dots	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead	Grayish/Black	Absent	Absent	Difffuse	Telangiectasia	Eccrine opening	Absent	Peri-follicular	Reticular
forehead	Grayish/Black	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
forehead	Grayish/Black	Dots	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Diffuse
forehead	Grayish/Black	Absent	Absent	Difffuse	Absent	Eccrine opening	Absent	Absent	Reticular
Forehead,malar, retroauricular	Violaceous	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Absent	Chinese letter arrangement
Forehead,malar, retroauricular	Brown	Absent	Globules	Absent	Telangiectasia	Eccrine opening	Absent	Absent	Diffuse
Forehead,malar, retroauricular	Violaceous	Dots	Absent	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Globules	Absent	Absent	Eccrine opening	Absent	Peri-follicular	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Absent	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Reticular
Forehead,malar, retroauricular	Violaceous	Dots	Globules	Absent	Absent	Eccrine opening	Absent	Absent	Chinese letter arrangement
Forehead,malar, retroauricular	Grayish/Black	Dots	Absent	Difffuse	Absent	Eccrine opening	Absent	Peri-follicular	Reticular

Epidermis	Epidermal Thickness	Upper dermal Lichenification	Duration of disease (months)	Number of Melanophages	Inflammatory infiltrate	Flatenning of rete ridges
Nornmal	Basal layer	None	4	6	Grade 1	Present
Acanthosis	2	Mild	5	7	Grade 1	Present
Normal	2	Mild	28	14	Grade 2	Present
Acanthosis	2	Mild	6	8	Grade 1	Present
Normal	2	Mild	8	9	Grade 1	Present
Acanthosis	Basal layer	Mild	6	6	Grade 1	Present
Acanthosis	2	Mild	5	11	Grade 2	Absent
Normal	2	None	4	10	Grade 2	Absent
Acanthosis	Basal layer	Moderate	4	6	Grade 1	Absent
Atrophy	>3	Mild	7	9	Grade 1	Absent
Normal	Basal layer	Mild	12	11	Grade 2	Present
Acanthosis	2	Mild	30	15	Grade 3	Present
Acanthosis	2	Mild	10	8	Grade 1	Absent
Normal	Basal layer	None	8	8	Grade 1	Present
Acanthosis	2	Mild	32	17	Grade 2	Present
Atrophy	2	Mild	12	10	Grade 2	Present
Normal	>3	Mild	18	14	Grade 3	Present
Acanthosis	Basal layer	Mild	10	8	Grade 1	Present
Atrophy	>3	Mild	16	15	Grade 2	Present
Acanthosis	2	None	7	11	Grade 2	Present
Atrophy	>3	Mild	24	18	Grade 2	Present
Acanthosis	Basal layer	Moderate	6	8	Grade 1	Present
Atrophy	>3	Mild	6	10	Grade 2	Present
Normal	2	Moderate	26	16	Grade 2	Absent
Atrophy	>3	Mild	11	8	Grade 1	Absent
Acanthosis	2	Mild	27	15	Grade 2	Absent
Atrophy	Basal layer	Mild	8	7	Grade 1	Absent
Acanthosis	2	Mild	10	9	Grade 1	Absent
Acanthosis	2	None	2	11	Grade 2	Absent
Normal	2	Mild	36	17	Grade 2	Absent
Acanthosis	2	Mild	8	10	Grade 2	Present
Acanthosis	2	Mild	38	21	Grade 3	Present
Atrophy	2	Mild	25	14	Grade 2	Present
Acanthosis	2	Mild	26	16	Grade 2	Present
Atrophy	2	Mild	5	11	Grade 2	Present
Normal	>3	Mild	8	11	Grade 2	Present
Acanthosis	2	None	35	16	Grade 2	Present
Acanthosis	2	Moderate	3	10	Grade 2	Present
Acanthosis	2	Mild	36	14	Grade 2	Present
Acanthosis	2	Moderate	44	15	Grade 2	Present
Atrophy	>3	None	45	16	Grade 2	Present
Normal	2	Mild	6	10	Grade 2	Absent
Atrophy	2	Moderate	24	17	Grade 2	Absent

Acanthosis	2	Mild	39	14	Grade 2	Absent
Atrophy	2	Mild	28	16	Grade 2	Absent
Acanthosis	2	Moderate	2	11	Grade 2	Present
Normal	2	None	32	18	Grade 2	Present
Atrophy	>3	Moderate	36	16	Grade 2	Present
Acanthosis	2	Mild	38	16	Grade 2	Present
Atrophy	2	Severe	44	18	Grade 2	Absent
Normal	2	None	8	10	Grade 2	Present
Atrophy	2	Severe	46	23	Grade 2	Present
Acanthosis	2	Mild	38	16	Grade 2	Absent
Normal	>3	Severe	38	14	Grade 2	Present
Atrophy	>3	Severe	28	15	Grade 2	Absent
Atrophy	Basal layer	Mild	44	22	Grade 3	Present