Dermatology Practical & Conceptual

Dermatoscopic Patterns in Vitiligo

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ABSTRACT Vitiligo is a chronic, acquired autoimmune pigmentary skin disease, most times it can be diagnosed clinically. Dermoscopy can confirm vitiligo in a non-invasive way. It is a diagnostic technique that visualizes sub-macroscopic morphological structures which correspond with specific histological structures. It detects subtle changes in the pigment pattern, evaluates vitiligo activity, attempts of re-pigmentation, leucotrichia, and differentiates it from other hypo pigmentary disorders. Most dermatoscopic clues used to assess vitiligo activity are found at the perifollicular level in the center and edge of the lesion. Perifollicular pigmentation is present in both active lesions and treated pigmented lesions with treatment. However, perifollicular depigmentation represents poor response, in treated lesions, and poor prognosis in untreated ones. The center of the lesion has reduced and/or absent pigment network, in active and stable lesions. If on dermoscopy the center of the lesion shows islands of pigment, erythema, or telangiectasias, re-pigmentation is suggested. At the periphery of the lesion, unstable vitiligo usually shows up as a diffuse border, trichrome pattern, micro-Koebner/comet tail phenomenon, satellite lesions, or a tapioca sago pattern. In stable lesions it is more frequent to find well defined or trichromic border. Pigmented lesions commonly present sharp borders and marginal or perilesional hyperpigmentation.

Introduction

Vitiligo is the most frequent pigmentation disorder, it affects 0.38 to 1% of the world population [1]. It prevails in both sexes equally and in 37% of cases, it begins before 12 years of age [1,2]. It is caused by the targeted destruction or loss of function of melanocytes. Its clinical features are achromic spots and poliosis, for it affects skin, hair, and mucosae. Vitiligo is diagnosed by clinical examination, no laboratory tests are required, however, skin biopsies may be useful to distinguish it from other pigmentary disorders [3].

Dermoscopy (or epiluminescence microscopy) is a diagnostic technique, which allows the visualization of sub-macroscopic morphological structures which correlate with specific histological structures [4]. Initially it was used for the study and screening of neoplastic pigmentary lesions, however, its use has gradually been extended to other dermatoses, including vitiligo. This diagnostic tool allows the detection of subtle changes in the pigmentary pattern that aid the non-invasive confirmation of vitiligo. Additionally, it assesses the disease activity and differentiates it from other hypo pigmentary disorders (piebaldism, tuberous sclerosis, pigment mosaicism, Waardenburg syndrome, etc).

Clinical Nomenclature of Vitiligo

According to its distribution, vitiligo can be classified in the following clinical forms: 1) Segmental: It has a rapid onset and can affect one, two, or multiple segments. 2) Non-segmental which is subdivided into six subtypes: a) Acrofacial, b) Generalized, c) Universal, d) Mucosal, e) Mixed and f) Less frequent variants. 3) Undetermined or unclassifiable: it does not meet the criteria of any of the previously mentioned types after 2 years of follow-up [2,5].

Some rare clinical presentations of vitiligo include two main types:

a. Follicular: primary depigmentation of the hair follicle and does not initially compromise the surrounding skin. Dermoscopy shows white hair, and depigmentation affects the cortex and follicular medulla, yet the surrounding skin is not affected. Leukotrichia precedes perifollicular depigmentation and eventually, achromic macules or leukoderma may appear [6].
b. Blue: the spots are bluish and asymptomatic; their coloration corresponds to the absence of epidermal melanocytes and the presence of dermal melanophages. In dermoscopy, it is possible to observe white spots with linear areas and multiple blue dots, reticular telangiectasias or homogeneous white areas without structure with multiple black-bluish and gray-bluish points in the form of an arch, linear, semicircular, and mottled that are explained by the arrangement of the melanophages in the dermis are appreciated [7,8].

The evolution of vitiligo can be:

a. Stable or inactive: there is no progression of the previous spots and no new spots in the last 12 months.
b. Unstable or active or progressive: the number, size, or both of existing vitiligo lesions increase in the last 12 months.
c. Repigmentation or regressive: the spots pigmented spontaneously or with treatment [2,5].

Decisions about the initiation, modification, and discontinuation of vitiligo treatment depend on its topography and evolution. The same patient may have stable, progressive, and regressive spots in separate locations at the same time. Clinical activity is the result of cellular and inflammatory mechanisms. Performing a skin biopsy to assess vitiligo activity histologically is impractical and increases the probability of complications such as the Koebner phenomenon, scarring, or infections.

Therefore, it is desirable to use a non-invasive diagnostic method to evaluate all macules. Dermoscopy is exempt from complications and allows the examination of all lesions. Additionally, the cellular dynamics of the disease can be observed. Despite dermoscopy has gained popularity in the study of vitiligo, clinical studies in this area are still scarce [9]. We will review the dermatoscopic patterns of vitiligo evolutionary stage.

Vitiligo and Dermoscopy

In the dermoscopic evaluation of normal skin, the pigment network corresponds to the pigmentation distributed along the epidermal ridges (homogeneous pigmented lines) interspersed with pale areas belonging to the papillary dermis [10,11]. Melanocytes are present in greater numbers on the slope of the ridges of the network compared to the tips of the ridges which emphasizes the reticular pattern. The net corresponds to the slope and the pale areas to the tips of the ridges of the network [9]. In contrast, in vitiligo the loss of melanocytes clears the pigment network beyond the pallor of the ridges, which produces a negative pigment network and creates different dermatoscopic patterns (Table 1 and Figure 1) [9-11].

Vitiligo lesions in their different evolutionary stages (stable, progression, or regression) can be difficult to distinguish clinically from other causes of hypopigmentation or depigmentation disorders. In 2004, Chuh and Zawar used dermatoscopy to study vitiligo and reported a pattern that was not present in other hypopigmentation diseases. In this

Table 1. Description of dermatoscopic structures with vitiligo.

Pattern/signs	Description
Unstable or progressive vitiligo	
Perifollicular pigmentation	Achromic spots at the pilosebaceous orifice present a homogeneous surrounding pigment with a tone like that of normal skin.
Reduced or absent pigment network	Whitening or loss of the pigment network that causes an absent pigment network.
Inverted pigment network	The white or depigmented pattern is in the form of a network with intermediate pigmentation.
Trichromic	Depigmented middle area of pseudo-scarring appearance, surrounding the main area is a faint yellowish-brown pigmentation and in the periphery the characteristic phototype of the patient (3 different shades).
Starburst	Extension or peripheral whitish linear projections in various directions.
Comet tail	Unidirectional linear extension or projection to an area adjacent to the initial vitiligo lesion.
micro-Koebner phenomenon	The appearance of isomorphic depigmented linear stretch marks distributed along the trauma line or around the main patch of vitiligo.
Tapioca sago or satellite lesions	Small white spots with no structure less than 1 mm in diameter are located around the main patch of vitiligo right on the perilesional skin that looks clinically normal.
Leukotrichia	White hairy hairs
Stable and pigmentary vitiligo	
Perifollicular depigmentation	Absence of surrounding pigment in the pilosebaceous orifice.
Perilesional/marginal hyperpigmentation	Darker pigmentation around achromic lesions.
Intra/perilesional erythema	Redness of the skin depigmented or on its margins.
Telangiectasias	Bright red dilated capillaries 1 to 4 mm in diameter; resemble spider veins.
Atrophy	The achromic spot that presents a decrease of one or several layers of the skin and its skin annexes causing thinned skin, folded and accompanied by telangiectasias.
Perifollicular pigmentation	The pilosebaceous orifice in the depigmented areas has homogeneous surrounding pigment and is darker than that of normal skin.

pattern, depigmentation with reservoirs of perifollicular pigment suggested that vitiligo was focally active or in repigmentation [12].

In a study of 176 patients with depigmentation, Meng et al found that residual perifollicular pigmentation (Figure 2) was more frequent in the progressive vitiligo group than in the stable vitiligo group (91.94% versus 62.86%, $P < 0.05$). Residual perifollicular pigmentation was absent in patients with depigmentation diseases other than vitiligo [13].

With dermoscopy, subtle changes in the pigment pattern can be detected, allowing an early and more accurate diagnosis [11]. In 30 patients with progressive vitiligo with less than one month of evolution, Thatte et al found that the most frequent dermatoscopic patterns were reduced or absent pigment network (Figure 2) and the inverted pigment network. Perifollicular and perilesional hyperpigmentation was uncommon. In the same study, a histopathological-dermatoscopic correlation was performed and the number of melanocytes was normal in 60% of patients, lower in 23.5%, and absent in 16.5%. Additionally, perivascular lymphocytic infiltrate and at the dermo-epidermal junction were observed in half of the cases [14]. However, this publication does not describe whether the determination of the number of melanocytes was made by immunohistochemistry. On the other hand, a normal or decreased number of melanocytes does not always reflect their proper functionality. In the initial stages of vitiligo, despite their misfunctions, the destruction of melanocytes does not always occur but, this is not observable in histopathology with hematoxylin and eosin.

In a study with one hundred cases, Wali et al found different dermatoscopic patterns corresponding with different evolutionary moments of the disease. In stable vitiligo, the most frequent were reticular, perifollicular, and marginal pigmentation while in progressive vitiligo trichromic patterns were found (Figure 2), in salt and pepper, starbursts, and comet tails. Patients already receiving treatment showed areas with erythema, telangiectasia, or atrophy, as well as patterns of perifollicular pigmentation and marginal reticular hyperpigmentation [11]. In addition to the patterns already indicated, progressive lesions may present perifollicular repigmentation points [15].

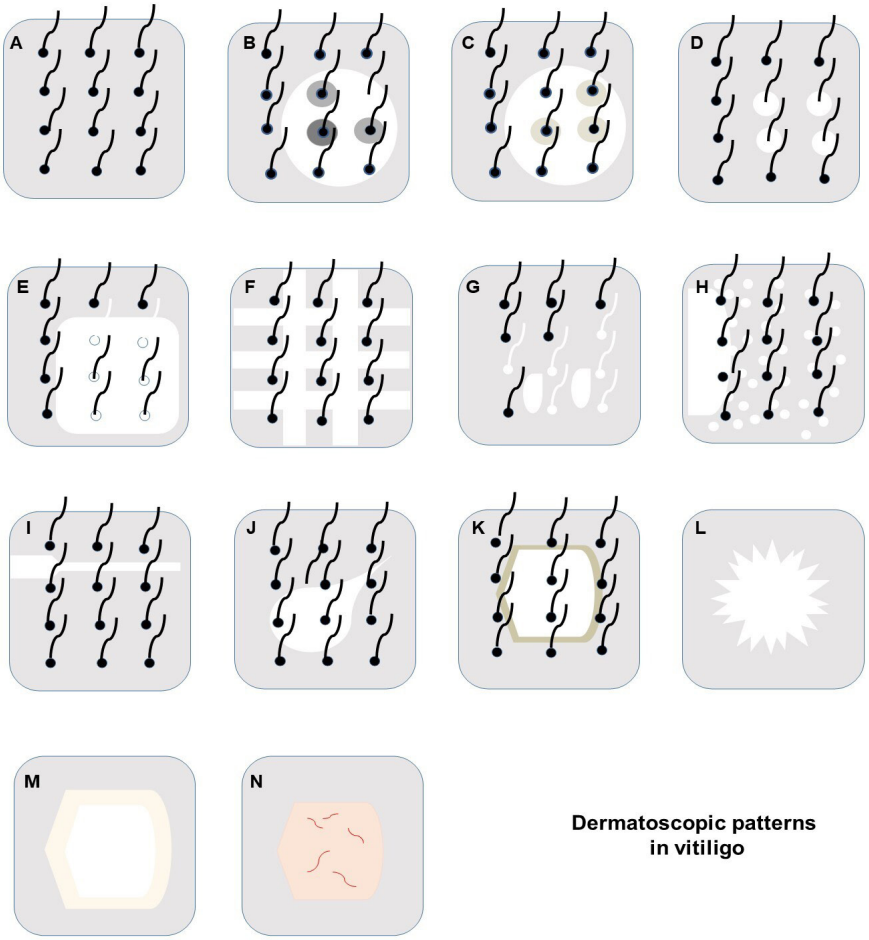

Figure 1. (A) Normal pigmentary pattern. (B) Perifollicular hyperpigmentation. (C) Perifollicular pigmentation. (D) Perifollicular depigmentation. (E) Reduced/absent network. (F) Reversed network. (G) Leukotrichia. (H) Tapioca sago appearance. (I) Micro-Koebner phenomenon. (J) Comet tail pattern. (K) Perilesional/marginal hyperpigmentation (L) Starburst pattern. (M) Trichrome pattern. (N) erythema with telangiectasia.

Jha et al also studied the association of dermatoscopic patterns with different evolutionary stages of the disease. They found that perifollicular pigment is an important dermatoscopic marker of vitiligo activity. Perifollicular pigmentation patterns and pigment network alteration (reduced, absent, or inverted) were more frequent in progressive vitiligo while perifollicular depigmentation and leukotrichia predominated in stable vitiligo. Leukotrichia (Figure 2) was found in both stable and regressive vitiligo. Treated vitiligo lesions presented perilesional hyperpigmentation and intra- or perilesional erythema with telangiectasias (Figure 2). In this study, a micro-Koebner microphenomenon was described for the first time that is characterized by forming isomorphic depigmented linear striations distributed along the trauma line or around the main patch of vitiligo. Morphologically it is different from that of a white comet tail composed of a unidirectional linear extension or projection to an area adjacent to the initial vitiligo lesion which appears in patients with progressive vitiligo. Both evoke koebnerization that is not detected by the naked eye highlighting the clinical value of dermatoscopic examination. Other dermatoscopic patterns observed in active vitiligo are named pattern in tapioca sago (Figure 2) which comprehends small white spots without a structure and less than 1 mm in diameter localized around the main vitiligo patch or on the perilesional skin that looks clinically normal [16].

In an additional study, Jha et al evaluated the dermatoscopic patterns that correlated with vitiligo activity. In progressive vitiligo, perifollicular pigmentation and alteration of the pigment network were most frequent, in stable vitiligo, perifollicular depigmentation predominated. Dermatoscopic patterns observed exclusively in progressive vitiligo lesions included: the starburst pattern, comet tail, and small pearly white globules, which were also observed in clinically normal perilesional skin of patients with progressive vitiligo. Leukotrichia was associated with refractoriness to treatment. Perilesional hyperpigmentation, intralesional or perilesional erythema, and telangiectasias were observed exclusively in patients responding to treatment. Their first publication also suggests that the loss or retention of perifollicular pigment is

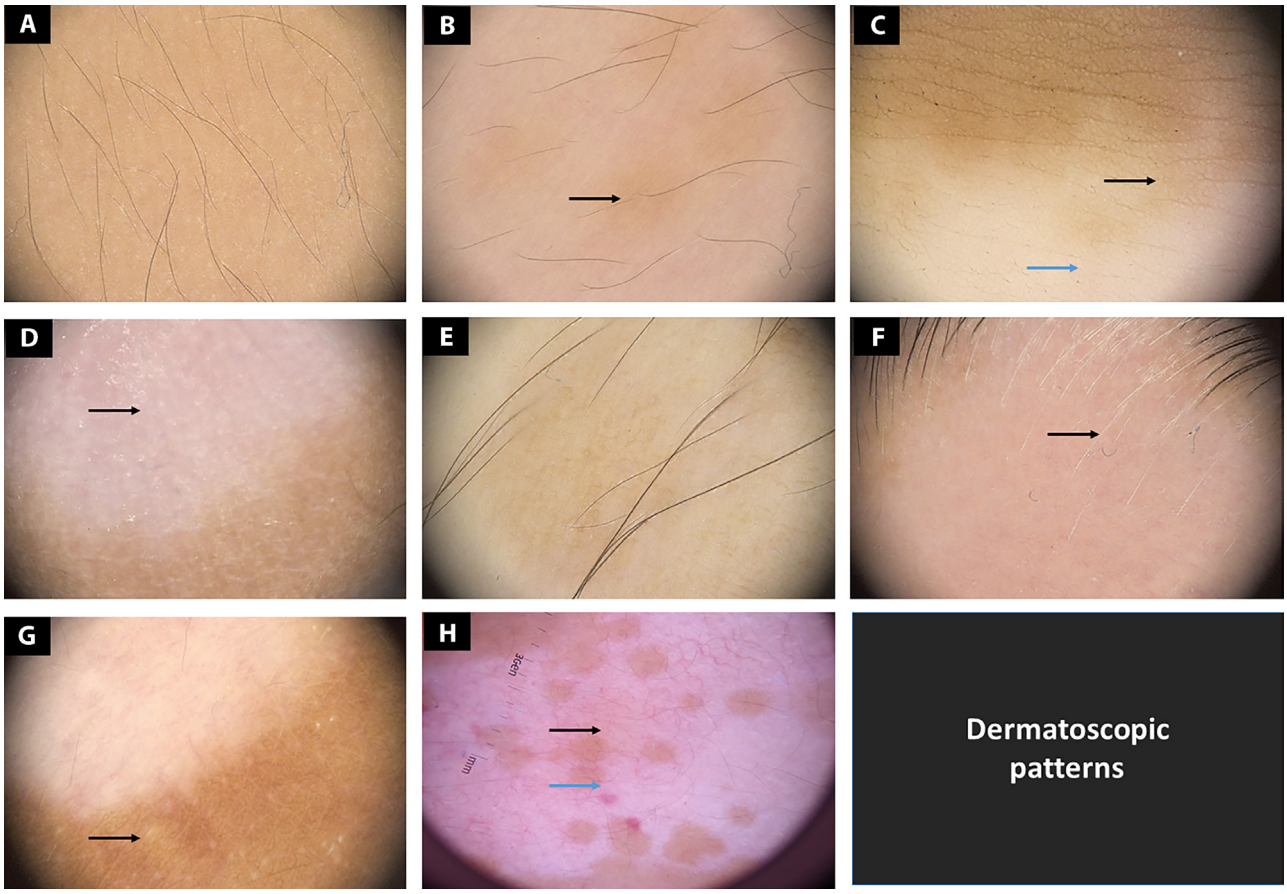

Figure 2. Representative dermoscopic images of vitiligo lesions (20x). (A) Normal skin reticulate pigmentary pattern. (B) Perifollicular pigmentation (black arrow). (C) Trichromic pattern, black arrow: intermediate pigmentation more faint yellowish brown, blue arrow: depigmented central area. (D) Absent network (black arrow). (E) Reduced network. (F) Leukotrichia (black arrow). (G) Tapioca sago (black arrow). (H) Erythema with telangiectasia (black arrow) accompanied by repigmentation isles (blue arrow).

an important dermatoscopic marker of vitiligo activity, perifollicular pigmentation suggests instability and perifollicular depigmentation is characteristic of stable disease [17].

In 2019, Nirmal et al evaluated the differences in dermatoscopic patterns between inactive and progressive vitiligo. The authors studied, in a cross-sectional and observational model, 131 lesions from eighty-five patients between 16 and 64 years of age using Wood light and dermatoscopy. Vitiligo activity was established using the vitiligo disease activity score (VIDA); lesions with a score of 0 to -1 were classified as stable (at least for 1 year or with spontaneous repigmentation) and those with a score of 1+ to 4+ as progressive (activity from 6 weeks or less) [18]. With Wood light, it was determined that the achromic lesions with well-defined boundaries were stable and that the progressive ones presented hypopigmentation, poorly defined boundaries, and trichromacy [9]. Dermatoscopically, stable vitiligo was characterized by the absence of satellite lesions and Koebner micro phenomena (sensitivity of 99.6% and 100% respectively). The presence of well-defined borders and cross-linked or absent pigment networks within the vitiligo spots were markers of stability (specificity of 100% and 96.8%). The retention of perifollicular pigment that Chuh and Zawar considered characteristic of stable vitiligo was less sensitive (66.5%) and was not significantly correlated with clinical stability in this study (P = 0.251). However, the specificity of perifollicular pigmentation was higher (81.4%) indicating that its absence is associated with unstable vitiligo [9].

So far, studies have described the different dermoscopic patterns at the edges of the lesions. The tapioca sago pattern, the satellite lesions and the "micro-confetti-like depigmentation" pattern mean depigmentation [9,16]. These perilesional macules are too small to be recognized with the naked eye. In addition, a poorly defined border also reflects lesion activity [9].

In vitiligo, melanocytes in hair follicles are one of the main sources of repigmentation. During dermatological examination with the naked eye, it is possible to observe the coloration of the hair shaft, whether it retains its pigment or becomes whitish (leukotrichia); segmental vitiligo is more associated with this sign than non-segmental vitiligo. For small hairs, dermoscopy is more useful than the naked eye to detect pigmentation. Additionally, dermoscopy differentiates white terminal hair (with greater length and thickness) from vellous hair vitiligo spots and other locations. Dong-Young et al observed that leukotrichia in segmental vitiligo may

insinuate a lack of response to medical treatment. Leukotrichia translates to the loss of melanocytes in the hair follicle, that is, the patient lacks a reservoir of melanocytes to repigment the achromic spots. It is advisable to institute initial treatment before most of the hairs located on the affected skin turn white [19].

Repigmentation-guiding Dermatoscopic Findings

Another useful aspect of dermatoscopy in vitiligo is the evaluation of the effectiveness of therapeutic interventions. ElGhareeb et al evaluated the dermatoscopic characteristics of vitiligo under systemic treatment. Patients who had repigmentation presented increased perifollicular, intralesional, and perilesional pigmentation, as well as a decrease in Koebner microphenomenon, a pattern in bursting stars and tapioca sago [20].

Errichetti et al evaluated the dermatoscopic characteristics of non-segmental vitiligo with repigmentation using narrowband UVB phototherapy. The lesions with better prognosis were on the face and those with perifollicular pigmentation. This pattern was twelve times more likely to repigment; peculiarly, there was no association with other dermatoscopic variables (leukotrichia, follicular red dots, and non-follicular pigmentation) [21].

Finally, Wang et al evaluated the efficacy of combined therapy with 308 nm excimer laser and topical tacrolimus in 147 patients. They found in progressive vitiligo lesions, before treatment, residual perifollicular pigmentation is more common (χ^2 = 23.180, P < 0.001) and perilesional hyperpigmentation is less common (χ^2=32.780, P < 0.001). Patients who repigmented dermoscopy revealed perifollicular repigmentation and early reservoirs of pigment at the edge of the lesion [22].

A consistent dermatoscopic hallmark in these studies is the appearance of the perifollicular pigment. The perifollicular zones are the main and initial source of repigmentation for the spots because melanocytic stem cells are at the hair bulb. Probably because their undifferentiated state prevents their autoimmune recognition, these cells are more resistant to T-cell attacks [23]. The existence of active dendritic melanocytes in the hair follicles of the repigmentation lesions has been observed with confocal reflectance microscopy [24].

Most of the studies we reviewed only reported descriptive statistics, therefore, it was not possible to assess any kind of association. Only a handful of works reported a risk-stratified analysis which could provide insight into the factors that influence clinical evolution, their association with dermatoscopic patterns, and the development of prognostic scores for this disease.

Conclusions

Dermoscopy is a non-invasive diagnostic tool that facilitates the clinical approach to vitiligo and allows the clinician to treat it according to its stage. The dermoscopic clues to assess vitiligo activity are found mainly at the perifollicular level, as depigmentation or pigmentation. In active and stable lesions, an absent pigment network is commonly seen in the center. Repigmentation presents as islands of pigment, erythema, or telangiectasias. Finally, at the periphery of the lesion, unstable vitiligo presents a diffuse or trichrome border, micro-Koebner/comet tail phenomenon, satellite lesions, or a tapioca *sago* pattern. Pigmented lesions have a sharp border and/or perilesional hyperpigmentation.

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