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Videodermoscopic Changes of the Hair in Vitiligo Lesions in Relation to Disease Duration

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ABSTRACT

Introduction: Vitiligo is an acquired disease of complex pathogenesis, in which the immunologic attack to the skin and hair follicle melanocytes leads to areas of depigmentation and leukotrichia, respectively.

Objectives: To study the dermoscopic features of the hair changes in vitiligo lesions in comparison to perilesional control areas and in relation to disease duration.

Methods: Forty-seven patients with both old and recent vitiligo lesions were included. Dermoscopic features of hair within the lesions were examined and compared to those in perilesional non depigmented skin of the same patient.

Results: Hair density ($P < 0.001$), terminal hair rate ($P = 0.011$), terminal to vellus hair ratio ($P = 0.029$) and mean hair shaft thickness ($P = 0.031$) were significantly decreased, whereas vellus hair rate ($P = 0.011$) was significantly increased in old vitiligo lesions compared to their respective control areas. The frequency of broken hair was significantly higher in old lesions ($P < 0.001$), while that of upright re-growing hair was significantly higher in recent lesions ($P = 0.016$).

Conclusions: Hair involvement in vitiligo lesions is not only limited to the development of leukotrichia. Other subtle changes in hair density, anagen and telogen hair rates, and mean hair thickness can be detected. These changes may serve as objective clues to the duration of the lesions.

Introduction

Vitiligo is an acquired chronic depigmenting autoimmune disease of the skin and hair; in which progressive destruction of melanocytes occur. Loss of melanocytes from the epidermis is the cause of leukoderma, and if the process extends to involve the active bulbar melanocytes, leukotrichia will develop [1]. In addition to the characteristic skin involvement in vitiligo, other associated manifestations including ocular and audiological findings have been described. [2-4].

Several theories have been put forward to explain the etiopathogenesis of vitiligo; among which autoimmunity is the leading one. The increased prevalence of several autoimmune diseases in vitiligo patients as well as their first degree relative has been reported, which suggests a general genetic predisposition to autoimmunity [4,5]. One of the commonly associated autoimmune diseases with vitiligo is alopecia areata (AA) [6]. Both diseases have common genetic risk factors and share certain similarities regarding their pathogenesis [7,8]. Several reports of colocalization of vitiligo and AA especially on the scalp exist in the literature [9-12], with skin biopsies exhibiting features of both diseases [12]. Moreover, relatively decreased hair density was previously reported in a depigmented area on the scalp that was also associated with histopathologic features suggestive of AA [13]. On revising the literature, no studies detailing the dermoscopic features of the hair in vitiligo lesions in different body areas could be found.

Objectives

The objective of this study was to explore the dermoscopic features of the hair in vitiligo lesions in different body sites and their relation to disease duration.

Methods

This study included 47 non segmental vitiligo patients of both sexes with more than one patch of vitiligo.

Exclusion criteria for lesion choice:

1. Lesions in areas anatomically known to have absent or sparse hair, eg palms, soles, mucous membranes, fingers and dorsum of the foot.
2. Scalp lesions, as it is difficult to evaluate vitiligo without shaving due to high hair density.
3. If all lesions in the same patient were of less than or more than two-year duration.
4. Segmental vitiligo.
5. Patients with a concomitant diagnosis of AA or with history of previously developing AA.

An informed consent was signed by each patient after the technical and scientific basis of the research project and the steps of the procedure were explained in details. The research was approved by the local Medical Ethics Committee (approval number: 0303878/14/03/2018).

Each patient was examined clinically and two vitiligo lesions (one old and one recent) were selected for examination; recent lesions were of a duration of less than two years and old lesions were of a duration of two or more years [14]. Lesions and surrounding control areas were photographed using a digital camera (Samsung ST66, 16 megapixels); photos were taken from a constant distance and under similar photographing conditions. The hair within the lesions was evaluated using the Medicam 1000 video-dermoscope (Fotofinder). Three shots were taken with the video-dermoscope (20X magnification) from the vitiligo lesion and three shots were taken from perilesional normally pigmented skin (control) after Wood's light examination was done to exclude the presence of subclinical vitiligo. The dermoscopic photos were evaluated by Trichoscale® Pro program (Fotofinder). The report of every photo included data about hair density, anagen and telogen hair rates, terminal and vellus hair rates and the mean hair shaft thickness. The means of the collected data from the reports of the three shots taken from every lesional and perilesional normally pigmented skin were determined. In addition, videodermoscopic photos of the studied lesions and perilesional control areas were evaluated for the presence of dermoscopic signs of alopecia areata such as black dots, tapering hair, broken hair, yellow dots, short vellus hair, circular (pigtail) hair, upright regrowing hair and Pohl-Pinkus constriction.

Statistical Analysis

The collected data was analyzed using the statistical package for Social Science (IBM SPSS Statistics for Windows, Version 22.0; IBM Corp.). Quantitative variables were tested for normality of distribution using the Kolmogorov-Smirnov test and were expressed as median and interquartile range (IQR). Categorical variables were expressed as frequencies and percentages. Paired nominal variables were compared using McNemar-Bowker test. Differences between paired continuous data (lesions and control areas) were tested using the Wilcoxon signed rank test. The strength of association between the two quantitative variables was assessed using Spearman method. A P value less than 0.05 was considered statistically significant.

Results

The age of the patients ranged from 7 to 55 years (mean: 26.38 ± 14.5 years). Twenty-four males (51%) and 23 females (49%) were enrolled in the study. The mean duration of the

included recent vitiligo lesions (< 2 years) was 6.28 ± 2.37 months while the mean duration of the included old vitiligo lesions (≥ 2 years) was 46.8 ± 18.72 months.

Hair Changes in Recent Lesions

In recent vitiligo lesions, anagen hair rate, anagen to telogen ratio, terminal hair rate and terminal to vellus ratio were lower than those in the respective control areas, but the difference was not significant (P = 0.352, 0.783, 0.603 and 0.656, respectively). Furthermore, hair density, telogen hair rate, and vellus hair rate were higher in recent vitiligo lesions compared to control areas, and the difference was also not significant (P = 0.151, 0.352 and 0.603, respectively) (Table 1).

Hair Changes in Old Lesions

In old vitiligo lesions, hair density, terminal hair rate, terminal to vellus ratio and mean hair thickness were significantly lower (P < 0.001, 0.011, 0.029 and 0.031, respectively), whereas vellus hair rate was significantly higher (p=0.011) than in the perilesional control areas. No significant difference between old patches and their control areas regarding anagen and telogen hair rates and anagen to telogen ratio was detected (P = 0.195, 0.195 and 0.357, respectively) (Table 2).

The decrease in hair density and thickness in old vitiligo lesions compared to their respective control sites is shown in Figure 1.

Dermoscopic Signs of the Hair in Vitiligo Lesions

In old lesions, the most commonly encountered sign was broken hair, which was seen in 51.1% of the patients, followed by pig tail hair in 31.9% of the patients. Meanwhile, tapering hair and Pohl-Pinkus constriction were the least commonly encountered signs, as each was detected in 8.5% of patients. In recent vitiligo patches, short vellus hair was detected in 31.9% of the patients followed by upright re-growing hair in 29.8% of the patients. The least commonly encountered sign was tapering hair that was detected in 4.3% of the patients. None of the recent vitiligo patches showed Pohl-Pinkus constriction. The presence of broken hair was significantly higher, whereas the presence of upright regrowing hair was significantly lower in old patches compared to recent patches (P <0.001 and 0.016, respectively) (Table 3). On comparing old vitiligo lesions to their respective controls, the frequency

Table 1. Comparison between hair changes in recent vitiligo patches and their respective control patches.

	Recent patches (N = 47)	Control of recent patches (N = 47)	P
Hair density (hair/cm^2)	52.00 (23.20 – 81.90)	45.40 (17.70 – 96.30)	0.151
Anagen hair rate (%)	51.70 (33.10 – 65.30)	53.50 (37.50 – 66.70)	0.352
Telogen hair rate (%)	48.30 (34.70 – 66.90)	46.50 (33.30 – 62.50)	0.352
Anagen to telogen ratio	1.07 (0.49 – 1.88)	1.15 (0.60 – 2.0)	0.783
Terminal hair rate (%)	5.60 (0.0 – 19.40)	6.70 (0.0 – 18.0)	0.603
Vellus hair rate (%)	94.40 (80.60 – 100.0)	93.30 (82.0 – 100.0)	0.603
Terminal to vellus ratio	0.06 (0.0 – 0.24)	0.07 (0.0 – 0.22)	0.656
Mean thickness (mm)	0.028 (0.026 – 0.031)	0.028 (0.025 – 0.031)	0.414

Data are expressed as median (interquartile range); P value for Wilcoxon Signed Ranks Test.

Table 2. Comparison between hair changes in old vitiligo patches and their respective control patches.

	Old patches (N = 47)	Control of old patches (N = 47)	p
Hair density (hair/cm^2)	33.20 (12.20 – 56.50)	78.60 (26.60 – 156.10)	< 0.001
Anagen hair rate (%)	48.10 (40.0 – 70.60)	61.60 (45.60 – 70.20)	0.195
Telogen hair rate (%)	51.90 (29.40 – 60.0)	38.40 (29.80 – 54.40)	0.195
Anagen to telogen ratio	0.92 (0.67 – 2.40)	1.60 (0.84 – 2.36)	0.357
Terminal hair rate (%)	10.30 (0.0 – 19.30)	17.10 (8.80 – 38.50)	0.011
Vellus hair rate (%)	89.70 (80.70 – 100.0)	82.90 (61.50 – 91.20)	0.011
Terminal to vellus ratio	0.11 (0.0 – 0.24)	0.21 (0.10 – 0.63)	0.029
Mean thickness (mm)	0.029 (0.025 – 0.031)	0.031 (0.029 – 0.035)	0.031

Data are expressed median (interquartile range); P value for Wilcoxon Signed Ranks Test

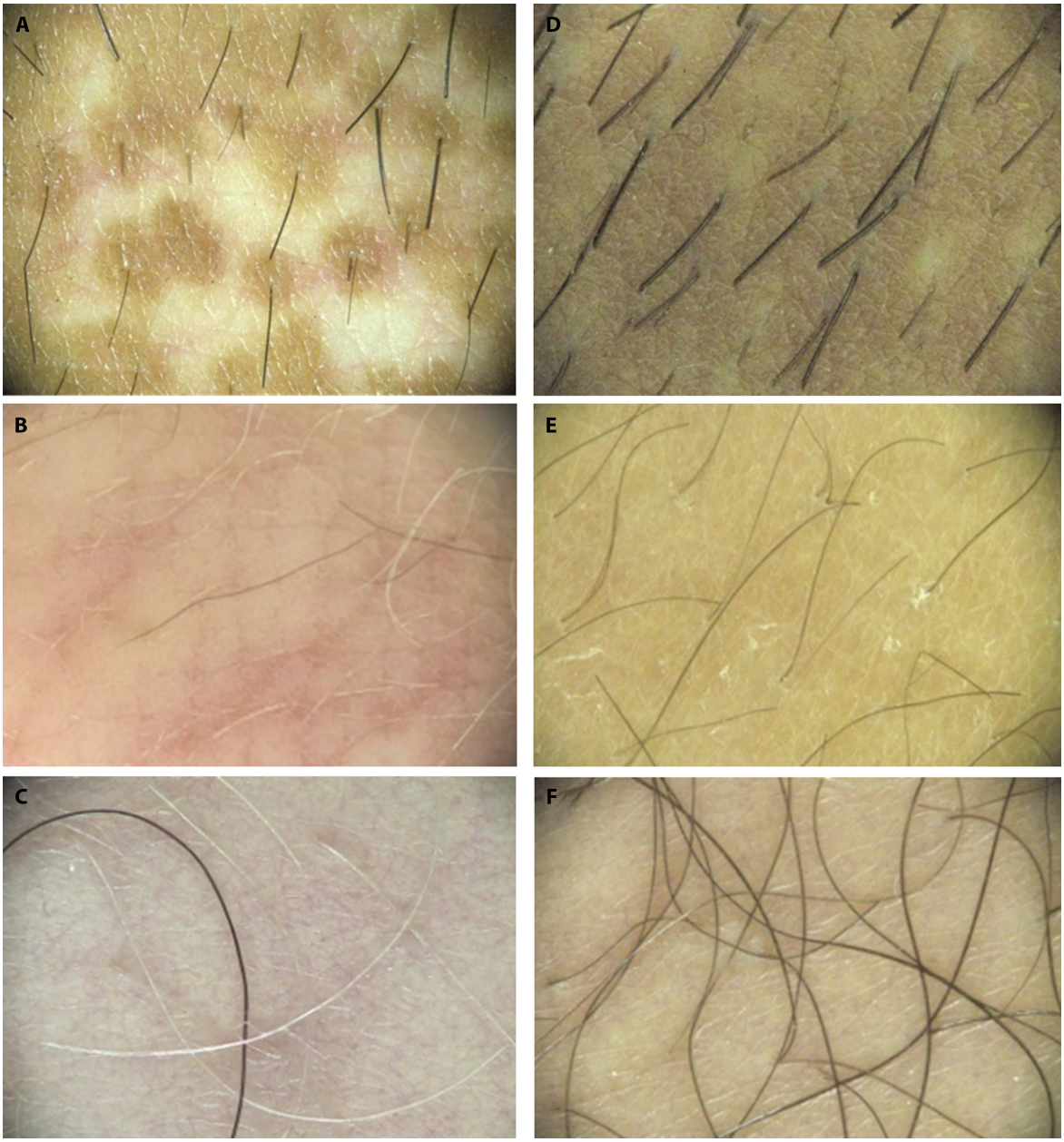

Figure 1. Decreased hair density and thickness in old vitiligo lesions compared to their respective control sites (magnification x20). (A-C) Videodermoscopic photos of the hair changes in the old vitiligo lesions. (D-F) Video-dermoscopic photos of respective control sites. (A, B, D and E on the nape, C and F on the leg).

Table 3. Comparison between old and recent vitiligo patches regarding dermoscopic features of alopecia areata incognita.

	Old patches (N =4 7)	Recent patches (N = 47)	P
Black dots, N (%)	6 (12.8%)	9 (19.1%)	0.250
Tapering hair, N (%)	4 (8.5%)	2 (4.3%)	0.500
Broken hair, N (%)	24 (51.1%)	8 (17.0%)	< 0.001
Yellow dots, N (%)	8 (17%)	6 (12.8%)	0.500
Short vellus hair, N (%)	11 (23.4%)	15 (31.9%)	0.125
Circular (pig tail) hair, N (%)	15 (31.9%)	11 (23.4%)	0.125
Upright re-growing hair, N (%)	7 (14.9%)	14 (29.8%)	0.016
Pohl-Pinkus constrictions, N (%)	4 (8.5%)	0 (0.0%)	0.125

P for McNemar-Bowker test

of broken hair, yellow dots and pig tail hair was significantly higher in the former ($P < 0.001$, 0.031, 0.004, respectively). Meanwhile, black dots and pig tail hair were significantly higher in recent vitiligo lesions than in control areas ($P = 0.004$ and 0.031, respectively).

Conclusions

In vitiligo, skin involvement usually precedes hair involvement. Nevertheless, lesions where leukotrichia developed prior to skin depigmentation have been reported, which was believed to result from preferential involvement of follicular melanocytes in such cases [15,16].

Several changes have been reported in association with vitiligo that include ocular, auditory and even nail changes [2,3,17]. Apart from the color change, little is known about the changes that could occur in the hair within vitiligo lesions. Reports of AA and vitiligo existing in the same individual are present [18], and several reports of co-localization of vitiligo and AA exist in the literature, most of which describe complete loss of hair in the depigmented patch with few white peripheral hairs. On histopathologic examination of the involved areas, features of vitiligo and AA were detected. Most of these cases were reported in young age and were mostly located on the scalp [9-12,19]. Walker et al. reported slight reduction in hair density within a localized area of poliosis on the scalp with depigmentation of the underlying skin in a male patient. Skin biopsies revealed features of both vitiligo and AA [13]. However, none of those reports studied the dermoscopic hair changes within the vitiligo lesions.

In the present study, the anagen-to-telogen and the terminal-to-vellus ratios were lower in both old and recent areas compared to their respective controls but the difference was significant only in the terminal-to-vellus ratio in old lesions. Furthermore, both old and recent vitiligo lesions exhibited dermoscopic findings such as yellow dots, broken hair, short vellus hair, upright regrowing hair and circular hair. These detected changes exhibit a certain degree of similarity to hair changes previously reported in alopecia areata incognita (AAI), which is characterized by the absence of a smooth bald surface, increased telogen hairs [20], yellow dots, upright regrowing hairs [21], black dots, short vellus hair, broken hair, and tapered hair [21-24]. AAI usually shows increased proportion of telogen and vellus hairs [25], resulting in decreased terminal-to-vellus and anagen-to-telogen ratios in patients compared to normal subjects [21,26,27].

The similarities between hair changes detected in the present study and those reported in AAI could be explained by the proposed immunologic link between vitiligo and AA; as both diseases are considered cell mediated autoimmune diseases in which disease induction, at least in part, has been attributed to an increase in cytotoxic CD8+NKG2D+ T cells together with over expression of interferon gamma (IFN-γ), with the involvement of the JAK-STAT signaling pathway [8]. In addition, it has been shown that the immunologic attack of CD8+ T-cells on follicular melanocytes can in some instances result in both depigmentation and hair loss [28]. Clinically, the fact that non-pigmented hairs are usually spared in alopecia areata, and regrowing hairs are initially depigmented, further links alopecia areata to melanocytes [29].

It has been proposed that autoreactive T-cells against melanogenesis-associated proteins are responsible for sparing white hairs in AA and for the transient regrowth of hypo-pigmented hairs. However, there is always an exception to the rule; as in some patients non-pigmented hairs were lost as well, and in others re-growing white hairs persisted. Furthermore, some patients never experience hair regrowth following the initial attack. These different outcomes suggest the existence of various antigens and various pathways involved that ultimately determine the fate of the immunologic attack on the hair follicle [30].

Unlike the findings of Walker et al. [13], hairs within the examined lesions were not exclusively depigmented, and in addition to decreased density, they also exhibited decreased thickness and decreased terminal to vellus ratio owing to an increase in vellus hair rate and a decrease in terminal hair rate. These changes, however, were only detected in old lesions. Unfortunately, the software program used in the current study digitally documents the essential hair parameters of all hairs in the lesion regardless of their color; accordingly, determining if the detected changes were influenced by hair color was not possible.

The role of disease duration in vitiligo has been previously studied and the presence of melanocytes in the hair follicles in vitiligo lesions was reported to be inversely correlated with disease duration [31]. In addition, shorter disease durations were associated with better treatment response to phototherapy [32]. In the present study, old and recent vitiligo lesions were compared with their respective controls. All significant videodermoscopic hair changes were exclusively seen in old vitiligo lesions.

These findings further point to the possible influence of disease duration in vitiligo. It seems that the insult to vitiligo skin is done through two waves with two different onsets, a first wave affecting epidermal melanocytes resulting in leukoderma, followed by a second wave affecting the hair leading to subtle hair changes, and if the insult of this wave is severe enough, leukotrichia can occur. The possibility that these hair changes could be the forerunners of leukotrichia remains to be determined.

To conclude, this study demonstrated that the hair involvement within vitiligo patches is not merely a color

change, and that these changes may serve as an objective clue to the duration of vitiligo lesions. The resemblance between the detected changes and some of the known features of AAI adds a new item to the list of similarities between AA and vitiligo. Accordingly, further research combining dermoscopic examination with histopathologic evaluation of the hair changes in vitiligo lesions and studying the relation of these changes to disease activity and severity is recommended.

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