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# Cicatricial alopecia: do clinical, trichoscopic, and histopathological diagnosis agree?

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## Abstract

**Introduction:** Cicatricial alopecia (CA) results from irreversible destruction and fibrosis of hair follicles. Trichoscopy offers a noninvasive method for diagnosis.
**Methods:** Thirty-two patients clinically diagnosed with CA were subjected to trichoscopy and histopathology assessment. The sensitivity and specificity of clinical and trichoscopic diagnoses were compared to histopathology.
**Results:** Thirty-two patients were clinically diagnosed as follows: 12 with discoid lupus erythematosus, four with lichen planopilaris (LPP), two with frontal fibrosing alopecia (FFA), three with folliculitis decalvans (FD), nine with central cicatricial centrifugal alopecia (CCCA), and two with long-term alopecia areata. Trichocopy revealed discoid lupus in 13 patients, LPP in nine, FFA in two, FD in three, central centrifugal alopecia in four, and pseudopelade in one. Histopathology confirmed discoid lupus in 13 patients, LPP in five, FFA in two, FD in three, CCCA in six, pseudopelade in two, and sarcoidosis in one. The sensitivity and specificity of clinical diagnosis were 69.2% and 84.2% in discoid lupus, 40.0% and 92.6% in LPP, 100.0% and 100.0% in FFA, 66.7% and 96.6% in FD, and 66.7% and 80.8% in central centrifugal alopecia. The sensitivity and specificity of trichoscopy were 84.6% and 89.5% in discoid lupus, 100.0% and 85.2% in LPP, 100.0% and 100.0% in FFA and FD, 66.7% and 100.0% in central centrifugal alopecia, and 50.0% and 100.0% in pseudopelade.
**Conclusions:** Trichoscopy can be equivalent to histopathology for diagnosing some cases of CA.

**Keywords:** cicatricial alopecia, dermoscopy, trichoscopy, histopathology

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## Introduction

Irreversible damage of the hair follicles with fibrosis is known as cicatricial alopecia (CA) with either primary or secondary causes (1–3). The North American Hair Research Society (NAHRS) divides primary CA into neutrophilic, lymphocytic, and mixed types in addition to a nonspecific end-stage group (4).

Diagnosis of CA is achieved via clinicopathological evaluation. However, definite diagnosis is challenging and sometimes inconclusive (5). The use of trichoscopy has been established in assisting the diagnosis of CA (6, 7).

This study evaluates the role of clinical and trichoscopic assessment in diagnosis of CA in relation to the cornerstone diagnostic tool, which is histopathology.

## Patients and methods

This prospective study included 32 patients with a clinical diagnosis of CA recruited from the Hair Clinic at the Faculty of Medicine, Alexandria University, Egypt from June 2018 to June 2019. Written informed consent was obtained from all participants. The study was approved by the local ethics committee in accordance with the 1975 Declaration of Helsinki.

Detailed history taking and examination were performed, and literature-based (8–12) provisional clinical diagnoses were made. Trichoscopic examination was performed using a handheld DermLite® DL4 dermatoscope (3Gen LLC, San Juan Capistrano, CA, USA). The polarized mode at tenfold magnification (dry and wet) was utilized. The dermatoscope was attached to an iPhone® 8 plus camera (Apple, Cupertino, CA, USA) through a connector, and images were captured.

Trichoscopic diagnosis was made based on typical signs and clues from the literature (6, 7, 13). General diagnostic features included absent follicular ostea, white irregular dots, and patches indicating a fibrotic process. Follicular plugging, branching blood vessels, or follicular red dots are known clues for diagnosis of discoid lupus erythematosus (DLE). Peripilar casts indicate a folliculocenteric inflammatory process in lichen planopilaris (LPP) along with blue-gray dots around hair follicles reflecting melanophage accumulation. Perifollicular erythema and lonely terminal hairs are suggestive of frontal fibrosing alopecia (FFA) with sometimes dystrophic eyebrow hair. A perifollicular white halo due to destruction of melanin in the outer root sheath ending in lamellar fibrosis can be encountered in central cicatricial centrifugal alopecia (CCCA). Pustules and yellowish scales are seen in folliculitis decalvans (FD) with tufted hair. Finally, the diagnosis of pseudopelade was made through exclusion and absent classical trichoscopic features of other CA forms.

Histopathology is the cornerstone for diagnosis. Two 4 mm punch scalp biopsies were taken from the active lesions and, if there were no signs of inflammation, biopsies were taken from the center of the patch. The first biopsy was processed for vertical sectioning and the other for horizontal sectioning: 5-micron sections were cut from formalin-fixed paraffin-embedded blocks, stained with H&E, and then examined by light microscopy. Typical features described in the literature were used for diagnosis (4, 5, 14).

A fibrous tract replacing hair follicles is an important feature of CA. In DLE, follicular epithelium showed vacuolar interface changes including interfollicular epidermis, epidermal atrophy, and follicular plugging. In addition, perifollicular, perieccrine,

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superficial, and deep perivascular lymphocytic infiltrate are integral features. Pigment incontinence and mucin deposition in the dermis are usually evident. In LPP, lichenoid band-like perifollicular lymphocytic infiltrate mainly affects the upper part of the follicles. The perifollicular infiltrate is more evident than in DLE. Similarly, FFA showed lichenoid perifollicular lymphocytic infiltrate in all cases. In CCCA, premature desquamation of the inner root sheath is found in some affected follicles, and concentric lamellar fibrosis is seen in some cases. In end-stage CA, residual lymphocytic infiltrate is rarely present with loss of sebaceous glands. In FD, the principal finding is intra- and interfollicular neutrophilic and lymphocytic infiltrate with fusion of infundibula.

### Statistical analysis

Data were entered into the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) (15). The Kolmogorov–Smirnov test was used to verify the normality of distribution of variables. Comparisons between groups for categorical variables were assessed using the chi-squared test (Monte Carlo). Specificity, sensitivity, positive predictive value (PPV), and negative predictive value (NPV) were assessed using histopathology as a reference standard.

## Results

The patients' ages ranged from 22 to 55 years and 22 (68.8%) were females and 10 (31.2%) were males. Clinically, the 32 patients had alopecic patches lacking follicular ostia. DLE was provisionally diagnosed in 12 patients based on presence of the following clinical features: adherent scales, follicular plugging, erythema, or areas of hypo- or hyperpigmentation. Only one case had musculoskeletal manifestations and positive anti-ds DNA. On trichoscopy, only 10 of them were found to have DLE, whereas two patients were found to have LPP and FD. On histopathology, only nine were shown to have DLE, whereas three were shown to have pseudopelade, sarcoidosis (Fig. 1), and FD (Table 1). Structureless yellow areas were described for the first time in 46.2% of DLE cases. It was also observed that the most common vascular pattern was linear (92.3% straight vessels and 46.2% serpentine vessels; Fig. 2).

LPP was provisionally diagnosed in four patients based on the clinical features of perifollicular inflammation and hyperkeratosis mainly in lesions located in the vertex. Only one patient had oral and cutaneous lesions. Trichoscopy supported the clinical diagnosis of LPP; however, histopathology confirmed the diagnosis in only two patients. The other two patients had CCCA and DLE (Figs. 3 and 4, Table 1). FFA was clinically diagnosed in two patients that were postmenopausal females with slow symmetric recession of the anterior hairline combined with loss of the lateral eyebrows and facial papules. The diagnosis was documented both trichoscopically and histopathologically in both patients (Table 1). Gray dots with a signet ring appearance were a new trichoscopic finding noted in both cases of FFA (Fig. 5).

CCCA was clinically diagnosed in nine patients with a smooth and shiny alopecic patch that mainly started on the central scalp and extended peripherally. Trichoscopy documented the specific

**Table 1 | Clinical versus trichoscopic versus histopathologic diagnosis.**

| Patient and sex | Clinical diagnosis | Trichoscopic diagnosis | Histopathologic diagnosis |
|---|---|---|---|
| 1 F | DLE | DLE | DLE |
| 2 F | DLE | DLE | DLE |
| 3 M | DLE | DLE | DLE |
| 4 M | AA | DLE | DLE |
| 5 F | AA | DLE | DLE |
| 6 F | DLE | DLE | DLE |
| 7 F | DLE | DLE | DLE |
| 8 F | DLE | DLE | DLE |
| 9 F | CCCA | DLE | DLE |
| 10 F | DLE | DLE | DLE |
| 11 M | DLE | DLE | DLE |
| 12 F | DLE | DLE | Pseudopelade |
| 13 M | DLE | DLE | Sarcoidosis |
| 14 F | CCCA | LPP | LPP |
| 15 F | CCCA | LPP | CCCA |
| 16 F | CCCA | LPP | LPP |
| 17 F | CCCA | LPP | LPP |
| 18 M | LPP | LPP | LPP |
| 19 F | DLE | LPP | DLE |
| 20 M | LPP | LPP | LPP |
| 21 F | LPP | LPP | CCCA |
| 22 M | LPP | LPP | DLE |
| 23 F | CCCA | CCCA | CCCA |
| 24 F | CCCA | CCCA | CCCA |
| 25 M | FD | CCCA | CCCA |
| 26 F | CCCA | CCCA | CCCA |
| 27 F | CCCA | Pseudopelade | Pseudopelade |
| 28 M | FD | FD | FD |
| 29 F | DLE | FD | FD |
| 30 M | FD | FD | FD |
| 31 F | FFA | FFA | FFA |
| 32 F | FFA | FFA | FFA |

F = female, M = male, DLE = discoid lupus erythematosus, AA = alopecia areata, LPP = lichen planopilaris, FFA = frontal fibrosing alopecia, FD = folliculitis decalvans, CCCA = central cicatricial centrifugal alopecia.

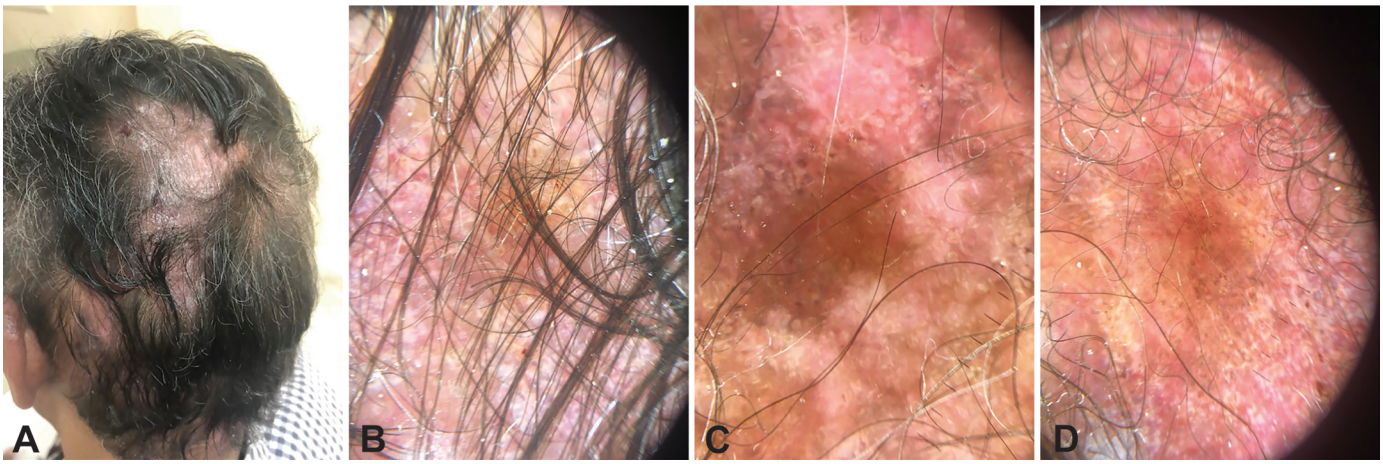


Figure 1 | A case diagnosed (a) clinically as discoid lupus erythematosus with patches of alopecia showing erythema and scaliness and (b) trichoscopically as discoid lupus erythematosus with red dots and white scales, (c) rosettes and brown discoloration, but (d) with an orange hue; however (e) histopathologic diagnosis was sarcoidosis with perifollicular granulomatous inflammation formed of histiocytes, multinucleated giant cells, and few lymphocytes (×200).

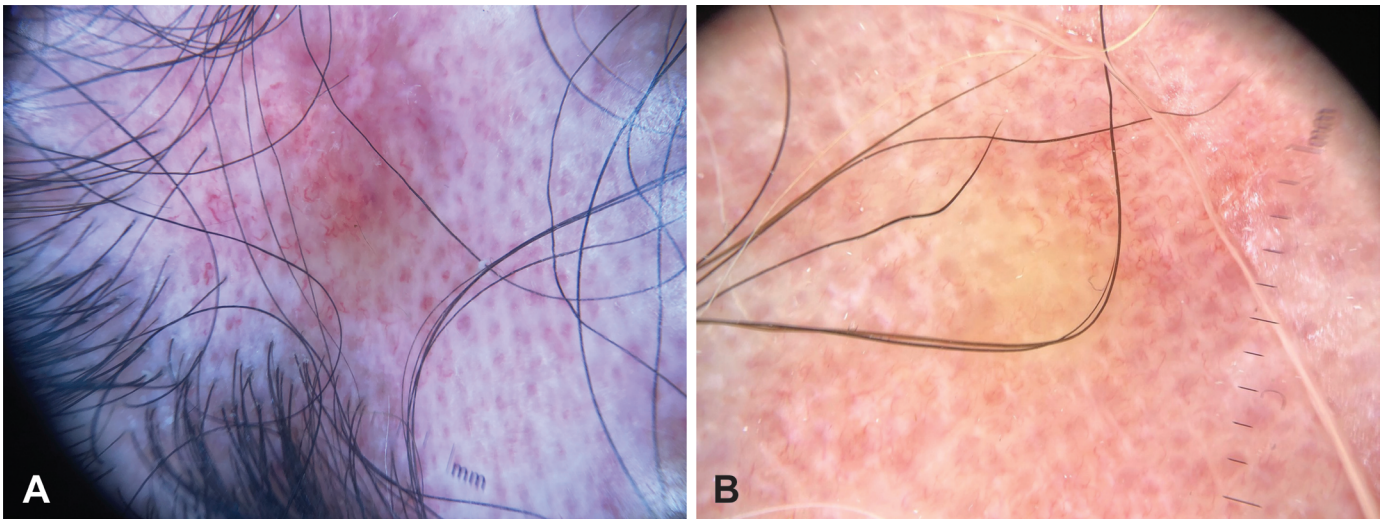


Figure 2 | Trichoscopy of discoid lupus erythematosus: the vascular pattern includes (a) thick and thin linear vessels (straight and serpentine), and (b) a structureless yellow area and linear vessels.

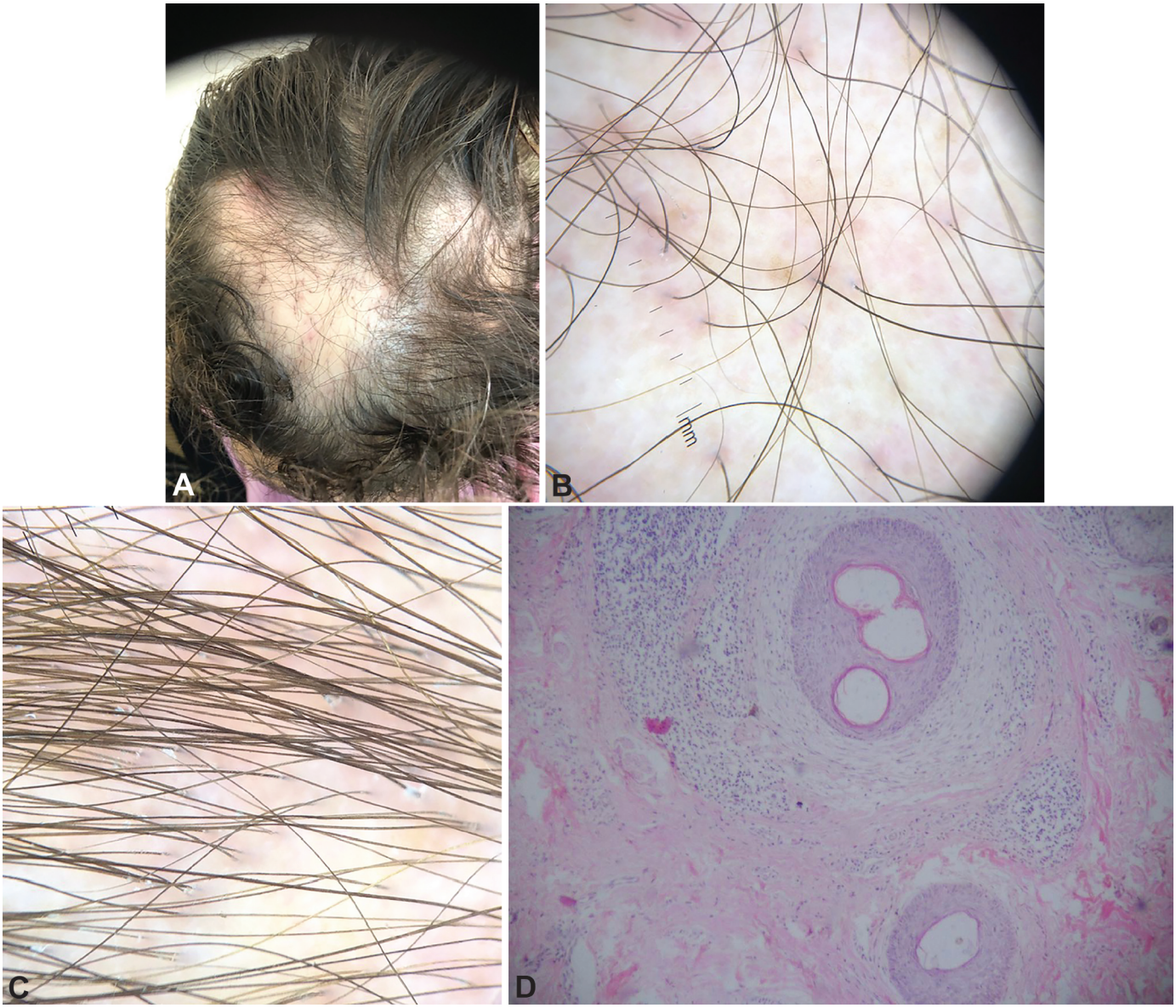


Figure 3 | A case diagnosed (a) clinically as lichen planopilaris and (b) trichoscopically as lichen planopilaris with perifollicular violaceous areas and (c) perifollicular white scales; however, (d) histopathological diagnosis was central cicatricial centrifugal alopecia with concentric lamellar fibrosis, eccentric epithelial atrophy, polytrichia, and mild lymphocytic perifollicular inflammation (H&E ×200).

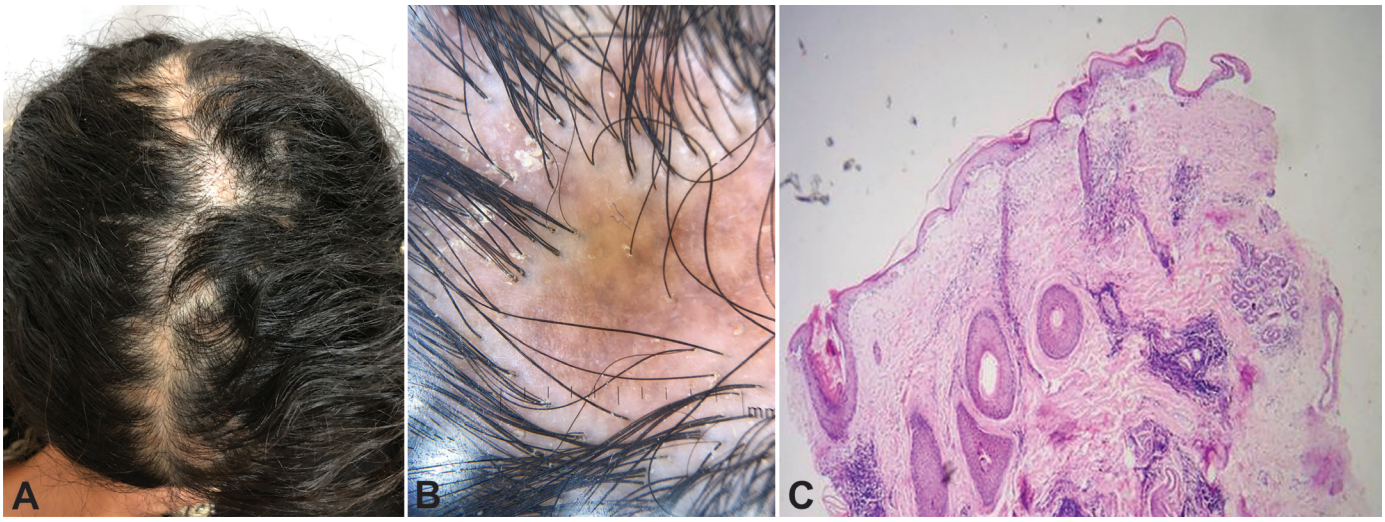


Figure 4 | A case diagnosed (a) clinically as lichen planopilaris and (b) trichoscopically as lichen planopilaris with perifollicular white scales, annular bluish-gray dots, and tufting; however, (c) histopathological diagnosis indicated discoid lupus erythematosus showing epidermal atrophy, follicular plugging, and periecrine lymphocytic infiltrate (H&E ×100).

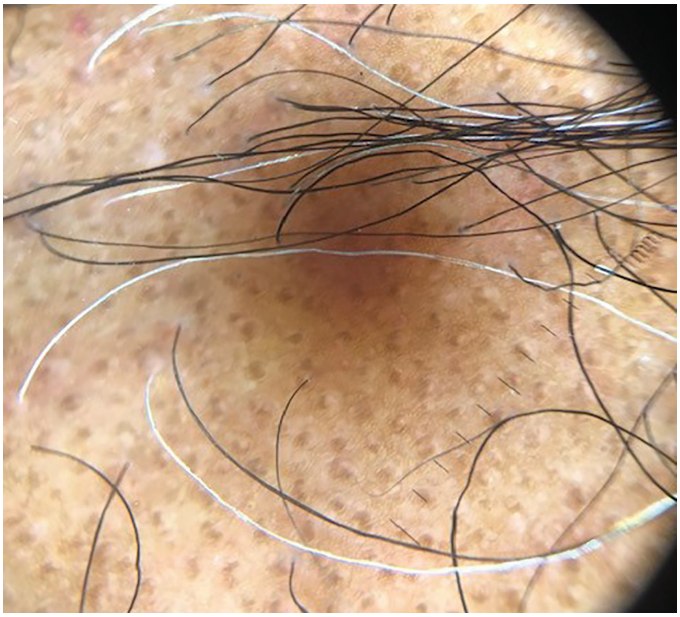

Figure 5 | Trichoscopy of frontal fibrosing alopecia showing gray dots with a signet ring appearance.

features in only three of these patients. Histopathology confirmed the diagnosis in only four of these patients, whereas among the other patients five were shown to have LPP, three were shown to have DLE, and the remaining two had pseudopelade. Three cases were diagnosed clinically as FD based on recurrent painful oozing, pustulation, and crustation in addition to polytrichia. Trichoscopy and histopathology confirmed the diagnosis in two cases, whereas the third one was confirmed as CCCA (Table 1). Two cases gave a clinical impression of long-term alopecia areata (AA), but with trichoscopy and histopathology they were confirmed as DLE.

Trichoscopic features are shown in Tables 2, 3, and 4, and histopathological features are shown in Table 4. Tables 6 and 7 present specificity, sensitivity, NPV, PPV, and accuracy for clinical and trichoscopic assessment of CA subtypes, taking into consideration histopathological diagnosis as a gold standard. Clinical assessment showed the highest specificity and sensitivity (100.0%) for FFA. On the other hand, trichoscopy had high specificity for diagnosing CCCA, FD, FFA, and pseudopelade (100.0%), and the highest sensitivity for FD, LPP, and FFA (100.0%).

The predominant inflammatory infiltrate was lymphocytic in 56% of cases. Absent or mild infiltrate was found in 25% of cases, neutrophilic infiltrate in 9%, and histiocytic infiltrate in 9%.

## Discussion

In this study, 32 patients were clinically diagnosed as CA with female predominance, in agreement with most other studies. This is in contrast to Kumar et al., who reported male predominance (5, 16, 17). This may be attributed to the greater tendency of females to consult for hair problems (14).

Taking into consideration that histopathology is the gold standard in diagnosis of CA, 31 (97%) cases were diagnosed as primary CA, in which preferential destruction of epithelial hair follicles with spared reticular dermis are the main features, and only one (3%) case was diagnosed as secondary CA due to sarcoidosis. This agrees with the findings by Kumar et al. (14). Early stages of primary CA can be classified according to type of infiltrate (14). Primary CA showed predominant lymphocytic infiltration, especially in DLE and LPP patients in 18 cases, followed by absent or mild inflammatory infiltration in eight cases mainly in CCCA and pseudopelade, whereas neutrophilic infiltration predominated in three cases of FD. Histiocytes and giant cells were noted in two cases of CCCA and, in the only secondary CA case, sarcoidosis as well. Villablanca et al. (18) found a similar frequency of predominance, whereas Kumar et al. (14) reported a lymphocytic predominance followed by neutrophilic predominance in cases of primary CA. Tan et al. (5) reported that the lymphocytic to neutrophilic CA ratio was 4:1, in line with data from Whiting et al. (19). It is generally postulated that a fibrous tract replacing the hair follicles is a common finding in cases of CA, especially in the late stage, which was encountered in 50% of our cases. Different specific histopathological features were encountered in this study and were supported by other studies (14, 16, 17).

Scalp sarcoidosis is a rare cutaneous manifestation of cutaneous sarcoidosis. The case here was clinically and trichoscopically misdiagnosed as DLE. The diagnosis was made based on histopathology, showing perifollicular sarcoidal granuloma formed of epithelioid histiocytes, and Langhans giant cells with scant lymphocytic infiltrate were detected. Some follicles were completely replaced by fibrous tissue, which is linked to a decline in the total number of terminal hairs. On reviewing the literature, scalp involvement is usually associated with other cutaneous effects,

**Table 2** | **Trichoscopic findings (follicular), *n* (%).**

| Follicular features | DLE (*n* = 13) | LPP (*n* = 9) | FFA (*n* = 2) | FD (*n* = 3) | CCCA (*n* = 4) | Pseudopelade (*n* = 1) | MC*p* |
|---|---|---|---|---|---|---|---|
| Absent follicular openings | 13 (100) | 9 (100) | 2 (100) | 3 (100) | 4 (100) | 1 (100) | – |
| Yellow dots | 9 (69) | 4 (44) | 0 (0) | 0 (0) | 0 (0) | 1 (100) | 0.023* |
| Large keratotic yellow dots | 4 (31) | 1 (11) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0.686 |
| Black dots | 6 (46) | 3 (33) | 2 (100) | 0 (0) | 0 (0) | 0 (0) | 0.131 |
| Fibrotic white dots | 13 (100) | 4 (44) | 1 (50) | 1 (33) | 4 (100) | 1 (100) | 0.004* |
| Targetoid bluish-gray dots | 7 (54) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0.026* |
| Red dots | 1 (8) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 1 (100) | 0.138 |
| Rosettes | 0 (0) | 1 (11) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0.591 |
| Gray dots with signet ring appearance | 0 (0) | 0 (0) | 2 (100) | 0 (0) | 0 (0) | 0 (0) | 0.002* |

* = statistically significant at $p \leq 0.05$.
MC = Monte Carlo, *p* = *p*-value for comparing between the different groups studied, DLE = discoid lupus erythematosus, LPP = lichen planopilaris, FFA = frontal fibrosing alopecia, FD = folliculitis decalvans, CCCA = central cicatricial centrifugal alopecia.

**Table 3** | **Trichoscopic findings (interfollicular and perifollicular), *n* (%).**

| Follicular features | DLE (*n* = 13) | LPP (*n* = 9) | FFA (*n* = 2) | FD (*n* = 3) | CCCA (*n* = 4) | Pseudopelade (*n* = 1) | MC*p* |
|---|---|---|---|---|---|---|---|
| White scales | 3 (23) | 8 (89) | 2 (100) | 2 (67) | 2 (50) | 0 (0) | 0.010* |
| Yellow scales | 0 (0) | 0 (0) | 0 (0) | 3 (100) | 0 (0) | 0 (0) | < 0.001* |
| Pinkish-white areas | 8 (62) | 2 (22) | 0 (0) | 2 (67) | 3 (75) | 0 (0) | 0.158 |
| Red areas | 6 (46) | 0 (0) | 1 (50) | 2 (67) | 0 (0) | 0 (0) | 0.033* |
| Structureless yellow areas | 6 (46) | 0 (0) | 0 (0) | 1 (33) | 1 (25) | 0 (0) | 0.149 |
| Violaceous areas | 1 (8) | 5 (56) | 0 (0) | 0 (0) | 1 (25) | 0 (0) | 0.112 |
| Scattered brown areas | 12 (92) | 2 (22) | 0 (0) | 1 (33) | 3 (75) | 1 (100) | < 0.001* |
| Polytrichia | 0 (0) | 1 (11) | 0 (0) | 3 (100) | 1 (25) | 0 (0) | 0.003* |
| Lonely hair | 0 (0) | 0 (0) | 2 (100) | 0 (0) | 0 (0) | 0 (0) | 0.002* |

* = statistically significant at $p \leq 0.05$.
MC = Monte Carlo, *p* = *p*-value for comparing between the different groups studied, DLE = discoid lupus erythematosus, LPP = lichen planopilaris, FFA = frontal fibrosing alopecia, FD = folliculitis decalvans, CCCA = central cicatricial centrifugal alopecia.

**Table 4** | **Trichoscopic findings (vascular patterns), *n* (%).**

| Vascular patterns | DLE (*n* = 13) | LPP (*n* = 9) | FFA (*n* = 2) | FD (*n* = 3) | CCCA (*n* = 4) | Pseudopelade (*n* = 1) | MC*p* |
|---|---|---|---|---|---|---|---|
| Comma vessels | 5 (39) | 0 (0) | 0 (0) | 1 (33) | 0 (0) | 0 (0) | 0.186 |
| Thick arborizing vessels | 1 (8) | 0 (0) | 0 (0) | 1 (33) | 0 (0) | 0 (0) | 0.500 |
| Thin arborizing vessels | 5 (39) | 1 (11) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0.484 |
| Straight linear | 12 (92) | 0 (0) | 0 (0) | 1 (33) | 0 (0) | 0 (0) | < 0.001* |
| Serpentine linear | 6 (46) | 0 (0) | 0 (0) | 1 (33) | 0 (0) | 0 (0) | 0.087 |
| Red spider in yellow dots | 3 (23) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0.542 |
| Perifollicular concentric hairpin vessels | 0 (0) | 0 (0) | 0 (0) | 2 (67) | 0 (0) | 0 (0) | 0.017* |

* = statistically significant at $p \leq 0.05$.
MC = Monte Carlo, *p* = *p*-value for comparing between the different groups studied, DLE = discoid lupus erythematosus, LPP = lichen planopilaris, FFA = frontal fibrosing alopecia, FD = folliculitis decalvans, CCCA = central cicatricial centrifugal alopecia.

**Table 5** | **Trichoscopic findings (follicular), *n* (%).**

| Feature | DLE (*n* = 13) | LPP (*n* = 5) | FFA (*n* = 2) | CCCA (*n* = 6) | Pseudopelade (*n* = 2) | FD (*n* = 3) | Sarcoidosis (*n* = 1) | MC*p* |
|---|---|---|---|---|---|---|---|---|
| Follicular plugging | 13 (100) | 3 (60) | 1 (50) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Epidermal atrophy | 11 (85) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Vacuolar alteration of basal cells | 13 (100) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Lichenoid perifollicular lymphocytic infiltrate | 0 (0) | 5 (100) | 2 (100) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Perifollicular and intrafollicular neutrophilic infiltrate | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 3 (100) | 0 (0) | < 0.001* |
| Perieccrine chronic inflammation | 13 (100) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Fibrous tracts replacing hair follicles | 6 (46) | 5 (100) | 1 (50) | 2 (33) | 2 (100) | 0 (0) | 0 (0) | 0.095 |
| Fusion of infundibulate (polytrichia) | 0 (0) | 0 (0) | 0 (0) | 2 (33) | 0 (0) | 3 (100) | 0 (0) | 0.001* |
| Involvement of interfollicular epidermis | 11 (85) | 2 (40) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Granuloma formation | 0 (0) | 0 (0) | 0 (0) | 1 (17) | 0 (0) | 0 (0) | 1 (100) | 0.051 |
| Pigmentary incontinence | 13 (100) | 5 (100) | 2 (100) | 0 (0) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |
| Concentric lamellar fibrosis | 4 (31) | 1 (20) | 1 (50) | 2 (33) | 0 (0) | 0 (0) | 0 (0) | 0.610 |
| Premature desquamation of IRS | 0 (0) | 0 (0) | 0 (0) | 6 (100) | 0 (0) | 0 (0) | 0 (0) | < 0.001* |

* = statistically significant at $p \leq 0.05$
MC = Monte Carlo, *p* = *p*-value for comparing between the different groups studied, DLE = discoid lupus erythematosus, LPP = lichen planopilaris, FFA = frontal fibrosing alopecia, FD = folliculitis decalvans, CCCA = central cicatricial centrifugal alopecia.

**Table 6** | **Relation between histopathology and clinical diagnosis ($n$ = 32).**

| Clinical | Histopathology | | Sensitivity (%) | Specificity (%) | PPV (%) | NPV (%) | Accuracy (%) |
|---|---|---|---|---|---|---|---|
| | Negative, $n$ (%) | Positive, $n$ (%) | | | | | |
| DLE | 19 | 13 | | | | | |
| Negative | 16 (84) | 4 (31) | 69.2 | 84.2 | 75.0 | 80.0 | 78.1 |
| Positive | 3 (16) | 9 (69) | | | | | |
| AA | 32 | 0 | | | | | |
| Negative | 30 (94) | 0 (0) | – | – | – | – | – |
| Positive | 2 (6) | 0 (0) | | | | | |
| CCCA | 26 | 6 | | | | | |
| Negative | 21 (81) | 2 (33) | 66.7 | 80.8 | 44.4 | 91.3 | 78.1 |
| Positive | 5 (19) | 4 (67) | | | | | |
| LPP | 27 | 5 | | | | | |
| Negative | 25 (93) | 3 (60) | 40.0 | 92.6 | 50.0 | 89.3 | 84.4 |
| Positive | 2 (7) | 2 (40) | | | | | |
| FD | 29 | 3 | | | | | |
| Negative | 28 (97) | 1 (33) | 66.7 | 96.6 | 66.7 | 96.6 | 93.8 |
| Positive | 1 (3) | 2 (67) | | | | | |
| FFA | 30 | 2 | | | | | |
| Negative | 30 (100) | 0 (0) | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Positive | 0 (0) | 2 (100) | | | | | |

DLE = discoid lupus erythematosus, AA = alopecia areata, CCCA = central cicatricial centrifugal alopecia, LPP = lichen planopilaris, FD = folliculitis decalvans, FFA = frontal fibrosing alopecia, PPV = positive predictive value, NPV = negative predictive value.

**Table 7** | **Relation between histopathology and trichoscopy diagnosis ($n$ = 32).**

| Trichoscopy | Histopathology | | Sensitivity (%) | Specificity (%) | PPV (%) | NPV (%) | Accuracy (%) |
|---|---|---|---|---|---|---|---|
| | Negative, $n$ (%) | Positive, $n$ (%) | | | | | |
| DLE | 19 | 13 | | | | | |
| Negative | 17 (90) | 2 (15) | 84.6 | 89.5 | 84.6 | 89.5 | 87.5 |
| Positive | 2 (11) | 11 (85) | | | | | |
| CCCA | 26 | 6 | | | | | |
| Negative | 26 (100) | 2 (33) | 66.7 | 100.0 | 100.0 | 92.9 | 93.8 |
| Positive | 0 (0) | 4 (67) | | | | | |
| LPP | 27 | 5 | | | | | |
| Negative | 23 (85) | 0 (0) | 100.0 | 85.2 | 55.6 | 100.0 | 87.5 |
| Positive | 4 (15) | 5 (100) | | | | | |
| FD | 29 | 3 | | | | | |
| Negative | 29 (100) | 0 (0) | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Positive | 0 (0) | 3 (100) | | | | | |
| FFA | 30 | 2 | | | | | |
| Negative | 30 (100) | 0 (0) | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |
| Positive | 0 (0) | 2 (100) | | | | | |
| Pseudopelade | 30 | 2 | | | | | |
| Negative | 30 (100) | 1 (50) | 50.0 | 100.0 | 100.0 | 96.8 | 96.9 |
| Positive | 0 (0) | 1 (50) | | | | | |

DLE = discoid lupus erythematosus, CCCA = central cicatricial centrifugal alopecia, LPP = lichen planopilaris, FD = folliculitis decalvans, FFA = frontal fibrosing alopecia, PPV = positive predictive value, NPV = negative predictive value.

which was not the case here (20). Radiological assessment was carried out for the patient, revealing pulmonary involvement. On reviewing the trichoscopic features and reevaluating the patient, a diffuse orange hue was the predominant clue, in agreement with Tores et al. (21).

Based on clinical assessment, taking histopathological diagnosis as the gold standard, the highest rate of misdiagnosis was reported in CCCA followed by DLE, whereas FFA showed the least reported misdiagnosis in this study. In a study by Qi et al. (22), LPP showed the highest rate of misdiagnosis, followed by pseudopelade, and the least for FD. Their cases were misdiagnosed as AA, androgenetic alopecia, and folliculitis, whereas in this study misdiagnosis applied to other forms of CA except for two cases of long-term AA. Of 12 patients that were provisionally diagnosed with DLE, nine were confirmed by histopathology in this study, and all clinically diagnosed DLE cases were confirmed by histopathological diagnosis in a previous report (5). Of 10 cases of LPP evaluated by Sorna Kumar et al. (23), eight cases were confirmed by histopathological examination and the other two cases were diagnosed as trichotillomania and morphea. In the same study, only one case of DLE was misdiagnosed clinically and was histopathologically confirmed as cutaneous T cell lymphoma. Of the four cases clinically diagnosed as LPP, only two patients were confirmed by biopsy in this study, whereas typical histopathological features of LPP were confirmed in cases evaluated by Tan et al. (5). Pseudopelade was clinically misdiagnosed as DLE and CCCA in this study and later reclassified by histopathological assessment. Pseudopelade is clinically easily missed because this was also reported in the study by Tan et al., in which nine patients were provisionally misdiagnosed as having androgenetic alopecia and AA; meanwhile, histopathological assessment revealed pseudopelade (5). In contrast, Villablanca et al. reported clinical overdiagnosis of pseudopelade. After histopathological examination, six cases initially diagnosed as pseudopelade were finally reclassified as other forms of CA. Hence, it was stated that pseudopelade is a challenging diagnosis (18). Similarly, Amato et al. reported changing the diagnosis of 66% of cases from pseudopelade to LPP and DLE after histopathological assessment (24). Pseudopelade is a term indicating a nonspecific scarring alopecia that has a noninflammatory gradual course. In fact, pseudopelade shows limited or even absent infil-

trate. Many types of CA may gradually burn out and turn into this category with undistinguishable histopathological or even clinical features (25). Opponents of this view postulate that pseudopelade is a separate type of CA or, alternatively, a form of end-stage of other scarring alopecias, such as DLE and LPP (17). Some authors assumed that 90% of pseudopelade results from LPP, whereas others have reported a lower percentage, only 15% (18, 25). Pseudopelade was the second most common type of CA in a previous report in contrast to the results of this study, in which CCCA is the second most frequent type of CA (18). In this study, a case clinically diagnosed as FD turned out to be CCCA with histopathology, as in Tan et al. (5). In contrast, Sorana Kumar et al. (23) reported histopathological confirmation of two clinically diagnosed cases of FD.

In a previous report, 38 cases of CA were diagnosed histopathologically as follows: two cases of LPP, two cases of FD, eight cases of DLE, a single case of scleroderma, 13 cases of pseudopelade, and 13 non-specific CA patients. The authors reassessed the diagnoses according to NAHRS and evaluated the thickness of the epidermal basement membrane and the dermal elastic system using PAS stains and Weigert, respectively. They reported 17 cases of DLE, four cases of LPP, 12 cases of pseudopelade, three cases of FD, one case of dissecting cellulitis, and individual cases of nonspecific alopecia. They concluded that further confirmation is possible by using those additional stains, which allowed definite diagnosis in 97.4% of their cases, especially in those misdiagnosed as non-specific CA (17). Villablanca et al. stated that one-third of CA patients cannot be diagnosed clinically, reflecting both diagnostic and therapeutic challenges. They emphasized the importance of biopsy in CA. This highlights our concept of histopathological assessment as the gold standard in approaching CA (18). This study tested the validity of clinical assessment of CA in relation to histopathological evaluation. The sensitivity and specificity were 69.2% and 84.2% for DLE, 66.7% and 80.8% for CCCA, 40.0% and 92.6% for LPP, 66.7% and 96.6% for FD, and 100.0% and 100.0% for FFA. Sorna Kumar et al. did not report a statistically significant correlation between clinical and histopathological assessment reported. They attributed this to a small sample size. However, they concluded that histopathology is the gold standard for diagnosing CA (23).

Trichoscopy is a non-invasive and simple tool for diagnosing CA and defining its subtype. Absent follicular openings were a pathognomonic feature in all cases of CA, distinguishing it from the non-cicatricial form, agreeing with previous studies (6, 26–28). It corresponds to hair follicle destruction with a specificity of 100% (29). Typical and unique trichoscopic features were encountered in the different CA subtypes. Although there is considerable contradictory information regarding their presence, follicular red dots were observed and even considered a distinctive finding confined to DLE cases (13, 30, 31). It is suggested that they are related to dilated vasculature surrounding dilated infundibula with extravasated red blood cells and overlying atrophic epidermis denoting viable follicles in an active stage with better prognosis (7). In contrasts to some studies, our results did not support arborizing vessels as a highly representative feature in DLE (29, 13, 32). A previous Korean study supported our view (27). On the other hand, straight linear vessels were documented in all our DLE cases, and hence these can be considered an integral feature in DLE. Gray dots with a signet ring were a unique finding for FFA in this study, and to the best of our knowledge this has not been described in previous literature. This can be explained by eccrine and follicular involvement in histopathology, suggesting that the dotted pattern and circles result from damage to those structures (33). A review of the literature showed a paucity of data for trichoscopy of scalp sarcoidosis, which is considered a rare entity. In this study, there were several nonspecific trichoscopic findings denoting CA. A unique sign was the orange hue documented in previous studies and explained by the presence of sarcoidal granuloma. Telangiectasia, another sign reported in previous studies, was not seen in this work, but there were pinkish white areas instead. While rosettes and brown dots surrounded by white haloes were two findings reported only in this study, Starace et al. described brown dilated follicular ostia and referred to these as a sign of activity. Nevertheless, there were interfollicular white scaling, black dots, and brown pigmentation (2, 20, 34). In this study, trichoscopic validity was tested for some forms of CA. The sensitivity and specificity of trichoscopy were 84.6% and 89.5% in DLE, 100.0% and 85.2% in LPP, 66.7% and 100.0% in CCCA, and 50.0% and 100.0% in pseudopelade. It should be noted that the highest sensitivity and specificity of trichoscopic features were achieved in FFA and FD. In accordance with literature, there is significant agreement between histopathology and trichoscopy in diagnosis of CA (7).

Good agreement between clinical and trichoscopic diagnosis was reported (Cohen's kappa = 0.824). There was 85% concordance between histopathological and trichoscopic diagnosis in the same study. The sensitivity and specificity of trichoscopy were 100.0% and 95.8% for LPP, 83.0% and 100.0% for pseudopelade, 100.0% and 95.8% for FD, and 100.0% and 100.0% for DLE (35). Thakur et al. (6) also concluded that 89% concordance was found between both tools in the diagnosis of CA.

## Conclusions

Histopathology is the gold standard for definite diagnosis of CA; however, trichoscopy is growing to a great extent, and in some cases it can approach the validity of biopsy, especially in cases of FFA, LPP, and FD. Gray dots with a signet ring appearance are a new trichoscopic sign in FFA. A linear vascular pattern is considered an important vascular sign in DLE.

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