A Study of the Histopathological Features of Alopecias on Transverse Sections of Scalp Biopsies

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Abstract
Background: Transverse sections of scalp biopsies are performed for the assessment of alopecias and considered advantageous over vertical sections. Aim: The aim was to study the histopathological features of alopecias on transverse sections of scalp biopsies. Methods: It was a descriptive study. Clinically confirmed cases of noncicatricial and cicatricial alopecias were subjected to 4 mm scalp biopsies, which were sectioned transversely and analyzed. Biopsies obtained from occipital region of androgenetic alopecia (AGA) cases were taken as controls. Results: Biopsies from 41 cases were assessed, including male and female AGA, alopecia areata (AA), trichotillomania, lichen planopilaris (LPP), discoid lupus erythematosus (DLE), and folliculitis decalvans (FD). Normal scalp (control) biopsies showed the median total number of hair follicles of 35 (32–37), anagen:telogen/catagen ratio of 17.5 (16.5–31), and terminal:vellus ratio of 15 (10.7–17.5). In AGA and AA, miniaturization and shift toward telogen and catagen hair were consistently observed. Peribulbar inflammation was seen in two-third of AA. Trichotillomania showed increased catagen hair and numerous pigment casts. In DLE, besides perifollicular inflammation, prominent peri-arrector pili and peri-eccrine inflammation were observed. Type of inflammatory infiltrate was similar in DLE and LPP (lymphocytic), whereas FD showed neutrophilic and plasma cell infiltrate, both around follicles and interstitially. Basal cell damage in the follicles and pigment incontinence were seen in majority of DLE and LPP patients. DLE also showed basement membrane thickening, mucin deposition, and telangiectasia. Reduction/absence of sebaceous glands and perifollicular fibrosis were observed in almost all cicatricial alopecias. Conclusion: Transverse sectioning may be a useful tool in the diagnosis of alopecias. Key Words: Alopecia, cicatricial, noncicatricial, transverse section

Introduction
Histopathology plays an important role in the diagnosis and assessment of disease activity in alopecia patients. Vertical sections that are routinely performed on skin biopsy specimens depict the entire skin thickness and are easy to section and process but do not provide enough information on quantitative and morphologic characteristics of hair follicles that are useful in making a diagnosis of alopecias. Transverse sections have become increasingly popular since their introduction in 1984 by Headington et al., as they circumvent the disadvantages of vertical sections. Present recommendation is to combine both transverse and vertical sections, and more recent methods (“Hovert” and “Tyler” techniques) have tried to achieve this through a single punch biopsy. From India, experience with transverse sectioning is limited in the published literature. We undertook this study to describe the histopathological features of alopecias on transverse sections of scalp punch biopsies.

Methods
It was a comparative study conducted between September 2012 and April 2014. The study was approved by our institute Ethics Committee (Reference Number IESC/T-224/01.06.2012). Scalp biopsies from clinically confirmed (confirmed by two experienced dermatologists based on clinical criteria laid down for these conditions) cicatricial and noncicatricial alopecia were taken. Due to difficulty in obtaining consent from healthy individuals for scalp biopsy, punch biopsies obtained from the unaffected occipital area of consenting male androgenetic alopecia (AGA) cases were taken as control.
Four millimeters punch biopsies, up to the depth of subcutaneous tissue, were obtained under local anesthesia from bald areas of noncicatricial alopecia and from active margins of cicatricial alopecia. Specimen was vertically embedded in paraffin, with the epidermal side facing upward. The specimen was progressively sectioned like a loaf of bread from the epidermal end downward. Sections from all levels (representative of all parts of a hair follicle) were taken. For each patient, eight slides were prepared from one biopsy sample, each slide containing four sections. All levels of hair follicle were represented in these 32 sections that were examined in detail. The histopathological features were evaluated by two dermatopathologists.

Data were analyzed using Stata 11.2 software (Stata Corporation, Texas, USA) and represented in frequency (percentage) and median (minimum-maximum). To assess the statistical significance of difference in frequency of histopathological features (categorical variables) between groups, Fisher’s exact test was used. For quantitative histopathological features (continuous variables), statistical significance was assessed among all groups using Kruskal–Wallis test, followed by Wilcoxon rank-sum test for intergroup analysis with Bonferroni correction.

**Results**

Scalp biopsy was obtained from 51 patients; of these, 10 patients were excluded from analysis due to oblique/tangential sectioning of the tissue block. Finally, data of 41 patients were analyzed; 21 of noncicatricial and 20 of cicatricial alopecia. The noncicatricial alopecia group included male (5 cases) and female AGA (4 cases), alopecia areata (AA, 6 cases), and trichotillomania (6 cases). Cicatricial alopecia group included lichen planopilaris (LPP, 7 cases), discoid lupus erythematosus (DLE, 7 cases), and folliculitis decalvans (FD, 6 cases). Biopsies from three male AGA cases (from unaffected occipital area) were included as controls.

The demographic and clinical profile of alopecia cases and controls is depicted in Table 1. Comparing the noncicatricial alopecia group and controls, there was no significant difference in the median number of hair follicles [Table 2]. Compared to controls, there was reduction in anagen-to-telogen/catagen (A:T/C) ratio in male and female AGA, AA, and trichotillomania [Figures 1-3]; however, it was not statistically significant. In male and female AGA, there was an increase in the number of telogen hair follicles, and in trichotillomania, there was an increase in catagen hair [Figure 3]. In AA, both catagen and telogen hair were increased [Figure 2].

There was significantly reduced terminal-to-vellus hair (T:V) ratio in male AGA (P<0.02) [Figure 1], female
AGA \((P<0.04)\), and AA [Figure 2] \((P<0.02)\) compared to control group. A significant reduction in T:V ratio in male \((P<0.006)\) and female AGA \((P<0.03)\) and AA \((P<0.01)\) compared to trichotillomania was also observed [Figure 3]. However, there was no significant difference in T:V ratio between AGA and AA.

The number of sebaceous glands was normal in all cases except two patients of AA (one each with ophiasis pattern and alopecia universalis), who had a reduced number of sebaceous glands. Fibrovascular stelae were observed in all AGA and AA biopsies, only one biopsy of trichotillomania and none in the control group. Peribulbar inflammation was only seen in AA (4 of 6 cases [67%], \(P<0.01\)) [Figure 2]. Pigment casts were observed in all trichotillomania cases (in anagen, catagen, and telogen hair follicles), four of six cases (67%) of AA (in catagen hair and fibrovascular stelae), one case of female AGA and in one control biopsy. In one trichotillomania patient, hemorrhage in follicular canal was observed. Perifollicular chronic inflammation was present in one case of trichotillomania. Mild perifollicular fibrosis was observed in one case each of female AGA (Ludwig grade 1) and AA (ophiasis pattern).

Among cicatricial alopecia patients, perifollicular inflammation was observed in almost all patients in the three groups [Figures 4-6] [Table 3]. Peri-arrector pili inflammation was seen more often in DLE (71%) compared to LPP (29%) and not seen in FD. Perieccrine inflammation was also seen more frequently in DLE (71%) compared to FD (17%) and not observed in LPP. Interstitial inflammation was more common in FD (67%) [Figure 6] as compared to DLE [Figure 5] (14%) and not seen in LPP [Figure 4]. A predominant lymphocytic infiltrate was noted in all LPP and DLE cases, whereas neutrophilic infiltrate was seen in FD (83%) [Figure 6]. A prominent plasma cell infiltrate was also noted in all FD cases and in 43% of DLE cases. Hair follicle rupture with foreign body granuloma formation was observed in one LPP and two FD cases [Figure 6].

Follicular basal cell damage was found more frequently in LPP (86%) and DLE (71%) compared to FD. Vascular degeneration of the basal cell layer was seen only in DLE (42.8%) [Figure 5]. Necrotic keratinocytes and colloid bodies were frequently seen in both LPP (57%) and DLE (57%). Pigment incontinence was seen in all

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### Table 1: Demographic profile and clinical features of alopecia cases and controls

| Clinical diagnosis          | Age (years) | Sex distribution | Disease duration (years) | Disease severity |
|----------------------------|-------------|------------------|--------------------------|-----------------|
| **Patients \((n=41)\)**     |             |                  |                          |                 |
| Noncicatricial alopecia \((n=21)\) | 26 (9-54)  | 22:20            | 4 (0.1-20)               |                 |
| AGA \((n=9)\)              | 25 (9-48)   | 0.7:1            | 4 (0.1-20)               |                 |
| Male AGA \((n=5)\)         | 27.5 (25-48)| 1.25:1          | 5 (3-20)                 |                 |
| Female AGA \((n=4)\)       | 43.5 (25-48)| 0:4             | 7.5 (4-20)               |                 |
| AA \((n=6)\)               | 16.5 (9-35) | 1:1              | 3 (0.4-10)               |                 |
| Trichotillomania \((n=6)\) | 19 (12-22)  | 1:5              | 3 (0.1-6)                |                 |
| Cicatricial alopecia \((n=20)\) | 30.5 (10-54) | 1.9:1         | 3 (0.2-20)               |                 |
| LPP \((n=7)\)              | 26 (14-47)  | 6:1              | 3 (0.3-12)               |                 |
| DLE \((n=7)\)              | 38 (22-51)  | 1:2.5            | 3 (0.2-8)                |                 |
| FD \((n=6)\)               | 23 (10-54)  | 5:1              | 4 (2-20)                 |                 |
| Controls \((n=3)\)         | 26 (25-30)  | 3:0              |                          |                 |

FD: Folliculitis decalvans, DLE: Discoid lupus erythematosus, LPP: Lichen planopilaris
LPP [Figure 4] and DLE patients and in one case of FD. Follicular basement membrane thickening was observed only in DLE patients (43%) [Figure 4].

Reduction/absence of sebaceous gland was a consistent finding in the cicatricial alopecia group. Dilated and ectatic capillaries were found predominantly in DLE (86%) compared to LPP (14%) and not seen in FD. Stromal mucin deposit was observed in two cases (29%) of DLE only.

Perifollicular fibrosis was observed in all LPP [Figure 4] and FD cases and only 28% of DLE cases. Ghost follicles (sites of hair follicles completely replaced by fibrosis) were more frequent in LPP (86%) compared to DLE (28%) and FD (17%).

Discussion

In our study, we analyzed the histopathological features of alopecias on transverse sections of scalp biopsies.
in 41 patients. In our study, the total number of hair follicles ($n=35$) from normal sites was found to be comparable to normal Iranian participants (Aslani et al.'s study – 36) and Caucasian Americans (Whiting’s study – 40 and Sperling’s study – 35.5). However, the number was higher compared to the Koreans (Lee et al. study – 16) and African-Americans (Sperling’s study – 21.5). The A:T/C ratio was comparable in all studies, ranging from 15:1 to 17.5:1.

Table 3: Comparison of histopathological features in patients with cicatricial alopecia

| Histopathological features | LPP ($n=7$) | DLE ($n=7$) | FD ($n=6$) | $P$ |
|----------------------------|------------|-------------|------------|-----|
| Compound follicles         | 0          | 1 (14.3) - 2-3 follicles | 1 (16.7) - 3-4 follicles | 0.9 |
| Perifollicular inflammation | 6 (85.7)   | 6 (85.7)    | 5 (83.3)   | 0.9 |
| Type of inflammation       |            |             |            |     |
| Lymphohistiocytic          | 6 (85.7)   | 6 (85.7)    | 6 (100)    | 0.99|
| Neutrophils                | 1 (14.3) - few | 1 (14.3) - few | 5 (83.3)   | 0.03|
| Eosinophils                | 0          | 1 (14.3) - few | 1 (16.7) - few | 0.9 |
| Plasma cells               | 2 (28.5) - few | 3 (42.9)    | 6 (100)    | 0.02|
| Follicular basal cell damage | 6 (85.7) | 5 (71.4)    | 1 (16.7)   | 0.2 |
| Vacular degeneration       | 0          | 3 (42.8)    | 0          | 0.08|
| Necrotic keratinocytes, colloid bodies | 4 (57.1) | 4 (57.1)    | 1 (16.7)   | 0.3 |
| Pigment incontinence       | 7 (100)    | 7 (100)     | 1 (16.7)   | 0.001|
| Basement membrane thickening | 0         | 3 (42.8)    | 0          | 0.08|
| Sebaceous gland reduced/absent in sections | 6 (85.7) | 6 (85.7)   | 6 (100)    | 0.9 |
| Peri-arrector pili inflammation | 2 (28.5) | 5 (71.4)    | 0          | 0.03|
| Perieccrine pili inflammation | 0          | 5 (71.4)    | 1 (16.7)   | 0.01|
| Intersitial inflammation   | 0          | 1 (14.3)    | 4 (66.7)   | 0.02|
| Dilated capillaries        | 1 (14.3)   | 6 (85.7)    | 0          | 0.01|
| Dermal edema               | 1 (14.3)   | 3 (42.8)    | 0          | 0.3 |
| Mucin                      | 0          | 2 (28.5)    | 0          | 0.3 |
| Perifolicular fibrosis     | 7 (100)    | 2 (28.5)    | 6 (100)    | 0.01|
| Ghost follicles            | 6 (85.7)   | 2 (28.5)    | 1 (16.7)   | 0.04|
| Interfollicular fibrosis   | 2 (28.5)   | 1 (14.3)    | 2 (33.3)   | 0.9 |
| Ruptured follice with foreign body granuloma reaction | 1 (14.3) | 0 | 2 (33.3) | 0.4 |

FD: Folliculitis decalvans, DLE: Discoid lupus erythematosus, LPP: Lichen planopilaris

In our male AGA cases, median T:V ratio was significantly reduced to 0.1 (0–1.3). In previous studies, the mean T:V ratio varied from 0.6 to 3.1 [Table 4]. The median A:T/C ratio in our study (3.6 [2.1–9.5]) was comparable to previous studies (2.3–6.1). In female AGA cases, median A:T/C ratio (4.15 [2–23]) and T:V ratio (1.0 [0.6–4.6]) were in accordance with previous studies with mean T:V ratio ranging from 1.5 to 2.9 and mean A:T/C ratio between 4.5 and 6.1. We observed greater miniaturization in male AGA (T:V ratio – 0.1) as compared to female AGA (T:V ratio – 1). This observation has also been made by other authors. The lower levels of 5-α reductase enzyme and androgen receptors with higher levels of aromatase enzyme expressed in the frontal scalp of women might explain the lesser severity of miniaturization in them.

The median A:T/C ratio (1.5 [0.8–7]) of our AA cases was comparable to studies by Horenstein et al. and Whiting, who observed ratios between 0.4 and 1.6. The T:V ratio was 0.8 (0–2.6) in our study with values ranging from 0.6 to 3.2 in previous studies. We observed greater miniaturization in extensive AA with ophiasis pattern (T:V ratio ranging from 0 to 0.4) compared to alopecia universalis (T:V ratio – 1.3) and localized AA (T:V ratio – 1.3, 2.6).

Based on quantitative parameters, no significant difference between AGA and AA was observed in our
study. Since pathogenic events in both the diseases ultimately result in premature termination of the anagen phase, the number of miniaturized and nonanagen hair counts can be expected to overlap in the two conditions. Other studies have also shown that it may be extremely difficult to differentiate between these two conditions based on quantitative assessment of hair follicles.\(^{[10]}\)

The median A:T/C ratio (3.4 [0.7–24]) and T:V ratio (8.6 [2.3–16.5]) of trichotillomania patients showed an increase in the number of nonminiaturized telogen and catagen hair, which was in agreement with previous studies.\(^{[10]}\) In the present study, a significant increase in catagen compared to telogen hair was seen, a feature characteristic of trichotillomania, and suggestive of a recent history of plucking in the area from which the biopsy was taken.

Multiple fibrovascular stelae were observed in all AGA and AA cases and also in one case of trichotillomania. This feature is observed as a result of miniaturization and an increase in the number of telogen/catagen hair follicles.

Perifollicular inflammation was seen in one case of trichotillomania only. In trichotillomania, rarely, foreign body granulomas to naked hair shafts or sometimes mild-to-moderate chronic inflammatory infiltrate can be seen.\(^{[10]}\) Role of inflammation in AGA is controversial with only a few studies supporting its significance in the pathogenesis of AGA. Whiting found perifollicular inflammation in 9% of normal participants and 36% of AGA.\(^{[5]}\) Aslani et al. observed this finding in 19% of AGA biopsies.\(^{[5]}\) Interestingly, Magro et al. found significant lymphocytic folliculitis on light microscopy, with immunoglobulin M and complement deposits on DIF, in a subset of their female AGA patients.\(^{[5]}\) They also experienced good clinical response with topical steroids and minocycline. However, we did not find any significant inflammatory infiltrate in our AGA patients.

Mild perifollicular fibrosis was observed in one case each of female AGA and AA in our study. Androgens have been found to directly stimulate not only dermal papillae cells but also interfollicular dermal fibroblasts to produce an increased amount of collagen. Furthermore, they indirectly augment fibrosis by inducing transforming growth factor-β 1 secretion from dermal papilla cells. Gross perifollicular fibrosis is not a usual feature of noncicatricial alopecia; however, in chronic longstanding AGA and AA, perifollicular fibrosis and follicular dropout have been described.\(^{[14]}\) Our AA case with perifollicular fibrosis had an ophiasis pattern of hair loss with near complete follicular miniaturization and reduced number of sebaceous glands. Normal sebaceous glands were present in all our noncicatricial alopecia cases except two patients of localized AA and one case of longstanding (10-year duration) alopecia universalis, in which they were reduced. Al-Zaid et al. evaluated sebaceous gland loss in alopecia by counting the number of hair follicles with or without associated sebaceous glands.\(^{[14]}\) They observed this feature in one case each of AA and AGA only.

In our study, a mild-to-moderate peribulbar lymphocytic inflammation was seen only in AA (4/6, 67%). Peckham et al. found peribulbar infiltrate in 84% (n=109) and Elston et al. in 44% (n=71) of AA cases.\(^{[17,18]}\) Jameel et al. observed this feature in 90% of cases with 1–6-month disease duration, in 71% with 6 months to 1 year of disease, and 66.6% cases with 1–5-year duration of alopecia.\(^{[19]}\) No correlation between the duration of alopecic patch and peribulbar infiltrate was observed in our study.

Pigment casts of varying shapes (twisted, dot-like, linear, and clumps) were observed in all cases of trichotillomania both within the epithelium and in hair canal. They were noted in anagen, catagen, and telogen hair follicles. Miteva et al. have recently described various morphological patterns of pigment casts in

| Study                        | Total hair count | Terminal: Vellus hair ratio | Anagen: Catagen/telogen ratio |
|------------------------------|-------------------|----------------------------|------------------------------|
| Aslani et al.\(^{[5]}\) (n=58) (0.9:1) | 29.1 30.1 29.6 | 3.1: 1 2.9:1 3:1 | 4.3: 1 4.5:1 4.3:1 |
| Horenstein and Bachelier\(^{[10]}\) (n=95) (1:5.7) | - - 25.5 | 2.0: 1 1.5:1 1.6:1 | 4.0: 1 5.8:1 5.4:1 |
| Whiting\(^{[6]}\) (n=106) (males) | 35 - 35 | 1.7: 1 - - | 5.3: 1 - - |
| Ekmekci et al.\(^{[11]}\) (n=40) (females) | - 31.7 31.7 | - 2.9:1 2.9:1 | - 6.1:1 - |
| Whiting et al.\(^{[6]}\) (n=120) (0.25:1) | - - - | 0.6-0.8: 1 1.7-1.9:1 | 2.3-3:1 5.7-6.1:1 |
| Current study (n=9) (1.25:1) | 32 23.5 25 | 0.1 1.0 0.7 | 3.6 4.15 3.5 |

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Table 4: Histopathological features of androgenetic alopecia patients in different studies
trichotillomania (zip sign and button sign), which were also noted by us.[20] Kulkarni et al. also noted pigment casts in 11 (44%) AA cases.[21] Pigment casts were also present in our AA cases, both in the catagen hair and follicular stelae. Miteva et al. had concluded in their study that the presence of pigment casts within miniaturized follicles and fibrovascular stelae were important clues to the diagnosis of AA.

We also noted pigment cast in one case of female AGA (telogen follicle) and in a control (in the catagen follicle). Hair biology studies have shown that matrix melanocytes undergo apoptosis during catagen, and the pigment not yet incorporated into the hair shaft is found degraded into the hair canal, from which it is extruded as melanin dust.[20] Our results suggest that pigment casts in catagen/telogen hair are suggestive of a diagnosis of either trichotillomania or a disease causing damage to the hair bulb.

In our cicatricial alopecia group, perifollicular inflammation was present in almost all patients. In FD, both perifollicular and interstitial infiltrate was noted. Periappendageal (around arrector pili and eccrine gland) and perivascular inflammation was also noted in DLE (71% cases). This observation is in accordance with previous studies which have also noted inflammatory infiltrate both around the superficial and deep vascular plexus and the periadnexal region, especially perieccrine location in DLE, while in LPP, the interfollicular epidermis is often spared, and the deep vascular plexus and other adnexal structures remain uninvolved.[14] The infiltrate was predominantly lymphocytic in LPP and DLE, neutrophilic in FD, and admixed with plasma cells in all FD and 43% of DLE cases. In FD, plasma cell infiltrate is a feature of chronicity of the disease; in fact, it may be an important clue to advanced disease.[22] Immunopathogenesis of cicatricial alopecia is far from clear at this point; however, Chiarini’s demonstration of a mixed Th1/2 response in FD compared to a strong CD8+ T cell response (Th1) in LPP might explain the more prominent plasma cell infiltrate in FD.[23]

Our findings of a more frequent basal cell damage in LPP and DLE, with prominent vacuolar degeneration in DLE and absence of significant basal cell damage in FD, were also in accordance with previous studies.[14] Reduction/absence of sebaceous glands was observed in nearly all cicatricial alopecia biopsies, with their preservation around few follicles in only one case each of DLE and LPP. Preservation of sebaceous glands in cicatricial alopecia may be due to focal involvement in early stages of disease, but this may not be the reason in our patients as the duration of alopecia in those with preserved sebaceous glands was 1 week in the DLE case and 3 months in LPP. In the other cases with absent glands, duration of disease ranged from 2 weeks to 6 months. Al-Zaid et al. found loss of sebaceous glands in 84.6% of LPP (n=13) and 100% of DLE and FD cases.[16]

In our study, perifollicular fibrosis was more often seen in LPP and FD compared to DLE, and ghost follicles were more frequent in LPP (86%) followed by DLE (29%) and FD (17%). Basement membrane thickening was noted in 43% of DLE cases. Thickened basement membrane is a classic and frequently reported histopathologic finding in DLE. In “burnt-out” lesions, this finding is preserved even though there is no inflammation.[14] Fabbri et al. found basement membrane thickening in 77% of patients in their study. Other features such as mucin deposition (28.5%), dilated capillaries (85.7%), and dermal edema (42.8%) were also observed in our DLE biopsies.

The limitation of the study was its small sample size. Good quality transverse sections are difficult to obtain as compared to vertical sectioning and require a trained technician in this field to consistently provide good transverse sections. Moreover, multiple levels of hair follicles have to be examined in different sections, which may be time-consuming. However, a representative section each from the infundibular (papillary dermis), isthmic, suprabulbar, and bulb at level of subcutaneous fat portion is most useful to make a diagnosis.

Conclusion

Transverse sectioning of scalp biopsies allows both quantitative and morphologic analysis of a large number of hair follicles. In our experience, on transverse sections, trichotillomania and FD have distinct histopathological features, while there are many overlapping features between AGA and AA and between LPP and DLE, which can make diagnosis difficult.

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Conflicts of interest
There are no conflicts of interest.

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