



Diagnostic Spectrum in Acute Diffuse Hair Shedding: A Clinical, Trichoscopic, Histopathological, and Immunohistochemical Study at Banadir Hospital, Mogadishu, Somalia

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Abstract

Background: Acute diffuse hair shedding in women is a diagnostically challenging presentation because telogen effluvium (TE), female pattern hair loss (FPHL), combined TE/FPHL, and diffuse forms of alopecia areata may present with overlapping clinical features. Trichoscopy, histopathology, and immunohistochemistry (IHC) may provide complementary diagnostic information, but their relative contributions in acute diffuse hair shedding remain incompletely characterized, particularly in African populations. The main objective of this study was to characterize the clinical and diagnostic spectrum of acute diffuse hair shedding in women and evaluate the complementary value of clinical assessment, trichoscopy, histopathology, and CD3 immunohistochemistry in differentiating TE, FPHL, and combined TE/FPHL, while assessing for features of alopecia areata incognita (AAI). **Methods:** A hospital-based cross-sectional study was conducted among 80 women aged ≥ 18 years who presented consecutively to Banadir Hospital, Mogadishu, Somalia, with acute diffuse hair shedding of ≤ 6 months' duration. Participants underwent standardized clinical assessment, hair-pull testing, trichoscopy using a FotoFinder Medicam 1000 videodermoscope, and scalp biopsy with histopathological and CD3 immunohistochemical assessment. Participants were classified into telogen effluvium, female pattern hair loss, or combined telogen effluvium/female pattern hair

loss based on predefined clinical and trichoscopic findings. Statistical analyses were performed using IBM SPSS Statistics version 27.0, with statistical significance set at $p < 0.05$. **Results:** The mean duration of hair shedding was 3.75 ± 1.0 months. Sudden onset occurred in 46 (57.5%) participants, while frontal and frontoparietal involvement accounted for 47.5% and 42.5%, respectively. TE was diagnosed in 40 (50.0%) participants, FPHL in 27 (33.8%), and combined TE/FPHL in 13 (16.3%). Significant differences were observed in several trichoscopic features. Hair-diameter diversity $\geq 20\%$ occurred in 5.0% of TE, 37.0% of FPHL, and 46.2% of combined cases ($p = 0.019$). Single-hair follicular units were present in 5.0%, 63.0%, and 46.2%, respectively ($p = 0.008$), while hair-shaft diameter differences occurred in 5.0%, 29.6%, and 61.5% ($p = 0.011$). Numerous frontal vellus hairs were observed in 0%, 37.0%, and 46.2% ($p = 0.009$), whereas short regrowing hairs occurred in 75.0% of TE, 0% of FPHL, and 76.9% of combined cases ($p = 0.004$). Peripilar signs were present in 22.2% of FPHL compared with none of the TE or combined cases ($p = 0.021$), while empty frontal follicles occurred in 29.6% of FPHL, 15.4% of combined cases, and none of the TE cases ($p = 0.031$). Multiple upright regrowing hairs occurred exclusively in the combined group (30.8%; $p = 0.024$). Quantitatively, mean hair-shaft diameter decreased progressively from TE to FPHL and combined TE/FPHL (64.8 ± 8.0 , 58.3 ± 8.0 , and 55.6 ± 4.0 μm , respectively; $p = 0.016$), while the number of thick hairs > 50 μm also differed significantly ($p = 0.041$). Histopathology showed predominantly mild lymphocytic infiltration without significant differences in inflammatory distribution among diagnostic groups. CD3 immunohistochemistry demonstrated T-lymphocytic infiltration across multiple tissue compartments but did not significantly discriminate the diagnostic groups. No participant fulfilled the predefined criteria for AAI. **Conclusion:** Acute diffuse hair shedding in women represents a heterogeneous diagnostic phenotype, with TE, FPHL, and combined TE/FPHL comprising the major diagnostic categories. Trichoscopy provided the strongest discriminatory information, particularly through assessment of hair-diameter variability, follicular-unit composition, frontal vellus hairs, regrowing hairs, and shaft caliber, despite similar overall hair density. Histopathology demonstrated predominantly mild and nonspecific inflammation, while CD3 immunohistochemistry did not significantly differentiate the diagnostic groups. An integrated clinical and trichoscopic approach, supplemented by histopathology and IHC when clinically indicated, may improve diagnostic characterization of women presenting with acute diffuse hair shedding.

Subject Areas

Clinical Medicine

Keywords

Telogen Effluvium, Female Pattern Hair Loss, Trichoscopy, Scalp Biopsy, CD3, Diffuse Hair Shedding, Alopecia

1. Background

Acute diffuse hair shedding is a common but diagnostically challenging presentation in dermatological practice. Unlike focal alopecias, in which the distribution and morphology of hair loss may provide relatively distinctive clinical clues, diffuse shedding can represent the phenotypic expression of several biologically distinct disorders. Telogen effluvium (TE) is classically regarded as the principal cause of acute diffuse non-scarring hair shedding and may follow physiological or psychological stressors, febrile illness, nutritional disturbance, rapid weight change, childbirth, systemic disease, medications, or other perturbations of the normal hair cycle [1]-[3]. Although acute TE is generally self-limiting, the clinical phenotype may overlap substantially with early androgenetic or pattern hair loss, diffuse alopecia areata, traction-related damage, nutritional or endocrine disorders, and the early stages of inflammatory or scarring alopecias. Consequently, the apparent simplicity of “diffuse hair shedding” can conceal considerable diagnostic heterogeneity [2] [4].

The diagnostic difficulty arises in part from the biology of the hair cycle. Hair follicles undergo tightly regulated transitions between anagen, catagen and telogen phases, and disturbances in follicular cycling can produce clinically similar patterns of increased shedding despite fundamentally different underlying mechanisms [2] [3]. In acute TE, a relatively large proportion of follicles prematurely enter the catagen/telogen pathway, resulting in increased shedding of telogen hairs. However, the magnitude and timing of this shift may vary according to the precipitating insult and individual susceptibility. Moreover, TE frequently coexists with other forms of alopecia, particularly pattern hair loss, making the distinction between an isolated shedding disorder and an underlying chronic follicular miniaturization process clinically important [2] [5]. This overlap can result in diagnostic uncertainty when assessment relies predominantly on history and conventional scalp examination.

A further challenge is the differentiation of acute TE from diffuse forms of alopecia areata and early pattern hair loss. These entities may present with apparently uniform reduction in hair density without conspicuous areas of complete baldness. Diffuse alopecia areata may lack the classic sharply demarcated patches and may show limited or absent clinically evident peribulbar inflammation, while pattern hair loss may initially manifest as increased shedding before more obvious follicular miniaturization becomes apparent [4] [6]. Histological studies have demonstrated that quantitative parameters such as the terminal-to-vellus follicle ratio and anagen-to-non-anagen ratio, together with qualitative features including peribulbar inflammation, pigment casts and follicular miniaturization, can assist in separating these conditions [6]. Thus, a purely clinical diagnosis may be insufficient in patients whose presentation lies at the boundary between major diagnostic categories.

Trichoscopy has substantially expanded the diagnostic assessment of hair disorders by permitting non-invasive visualization of follicular and interfollicular

structures that cannot be adequately appreciated by routine examination. Characteristic findings can assist in distinguishing non-scarring from scarring alopecia and can provide additional evidence for conditions such as alopecia areata and pattern hair loss [7] [8]. Importantly, trichoscopy may identify subtle abnormalities before they become clinically conspicuous and can guide the selection of an appropriate site for scalp biopsy when histopathological examination is required [7]. Nevertheless, its diagnostic contribution in acute TE is more complex because the trichoscopic phenotype may be relatively nonspecific. Reduced hair density, empty follicular openings and short regrowing hairs may occur, but these findings can overlap with other diffuse non-scarring alopecias [8] [9]. Therefore, although trichoscopy can increase diagnostic confidence, it should be interpreted in conjunction with clinical history and examination rather than considered an isolated diagnostic endpoint.

Histopathological examination remains particularly valuable when the clinical and trichoscopic findings do not adequately distinguish between competing diagnoses. Appropriate scalp biopsy and standardized histological processing permit assessment of follicular density, follicular cycling, terminal-to-vellus ratios, inflammation, fibrosis, pigment casts and other architectural abnormalities [10] [11]. In non-scarring alopecias, however, histological interpretation can itself be challenging. The findings of TE may be subtle or nonspecific, and diagnostic accuracy is strongly dependent on adequate tissue sampling, correct orientation, appropriate sectioning and integration of pathological findings with the clinical phenotype [10] [11]. Recent evidence further emphasizes that follicular counts and ratios can contribute to the distinction among TE, alopecia areata and androgenetic alopecia, but these quantitative measures should be interpreted alongside disease-specific morphological features rather than in isolation [11].

Immunohistochemistry (IHC) provides an additional level of tissue characterization when conventional histopathology is insufficient to resolve diagnostically overlapping alopecias. This is particularly relevant to disorders in which immune-mediated follicular injury contributes to hair loss. Alopecia areata, for example, is associated with inflammatory infiltration around and within the hair follicle, with T-lymphocyte populations playing an important role in disease pathogenesis [12] [13]. Immunophenotypic characterization using markers such as CD3, CD4 and CD8 has demonstrated potential utility in distinguishing alopecia areata from androgenetic alopecia, particularly in cases where conventional histological findings are equivocal [13] [14]. Other immunohistochemical markers have also been investigated in relation to follicular proliferation, differentiation and immune privilege, highlighting the potential of IHC to provide biological information beyond routine morphological examination [15] [16]. Nevertheless, the incremental diagnostic value of IHC in patients presenting specifically with acute diffuse hair shedding remains incompletely characterized, particularly outside highly specialized dermatopathology settings.

The diagnostic problem is therefore not simply the identification of excessive

hair shedding, but the accurate determination of the pathological process responsible for that shedding. A stepwise diagnostic strategy integrating clinical assessment, trichoscopy and histopathology has been advocated because the modalities provide complementary rather than interchangeable information [4] [7] [10]. Clinical examination establishes the temporal course, distribution and potential precipitating factors; trichoscopy provides a non-invasive assessment of follicular and scalp morphology; histopathology evaluates follicular architecture and inflammatory or degenerative changes; and IHC can further characterize cellular or immunological components when conventional morphology is inconclusive [10] [16]. Such multimodal assessment is particularly relevant where several forms of diffuse alopecia have overlapping clinical appearances.

Despite advances in hair-disorder diagnostics, evidence describing the integration of these modalities in African populations remains comparatively limited. This represents an important knowledge gap because diagnostic findings may be influenced by differences in hair morphology, grooming practices, environmental exposures, nutritional status, comorbidity profiles, healthcare-seeking patterns and access to specialized dermatological and dermatopathological services. Furthermore, evidence generated predominantly in European, North American and Asian populations may not necessarily capture the full clinical and pathological spectrum encountered in African dermatology practice. Establishing locally derived clinicopathological evidence is therefore important for improving diagnostic algorithms and avoiding both underdiagnosis and inappropriate treatment.

Somalia provides a particularly relevant setting in which to investigate this diagnostic problem. In routine clinical practice, patients presenting with acute diffuse hair shedding may initially receive a presumptive diagnosis based on clinical examination alone, despite the potential overlap among TE, pattern hair loss, alopecia areata and other causes of diffuse alopecia. A systematic evaluation combining clinical characterization with trichoscopy, histopathology and IHC has the potential to identify the extent to which these modalities converge or diverge in individual patients. Such an approach may also reveal diagnostic patterns that are not apparent from any single modality and could provide evidence for a more structured diagnostic pathway applicable to resource-constrained dermatological settings.

Accordingly, the present study at Banadir Hospital, Mogadishu, Somalia, investigates acute diffuse hair shedding through an integrated clinical, trichoscopic, histopathological and immunohistochemical framework. By correlating the clinical phenotype with trichoscopic characteristics and tissue-level morphological and immunophenotypic findings, the study seeks to clarify the diagnostic spectrum underlying acute diffuse hair shedding and to determine the complementary value of each diagnostic modality. This multimodal approach is intended not merely to describe hair-shedding patterns, but to address the central diagnostic spectrum of distinguishing clinically overlapping disorders and establishing a more objective basis for diagnosis in patients presenting with acute diffuse hair loss.

2. Materials and Methods

A hospital-based cross-sectional study was conducted among 80 women aged ≥ 18 years presenting to Banadir Hospital, Mogadishu, Somalia, with acute diffuse hair shedding of ≤ 6 months' duration. Women presenting to Banadir Hospital during the study period were consecutively assessed for eligibility, with the numbers screened, excluded, declined participation, and finally enrolled recorded according to the predefined eligibility criteria. Participants were eligible if they provided written informed consent and agreed to clinical examination, standardized scalp photography, trichoscopy, and scalp biopsy; women with localized/patchy alopecia, loose anagen hair syndrome, anagen effluvium, scalp infection or inflammatory disease, systemic autoimmune or other systemic conditions known to cause diffuse hair loss, recent topical or systemic hair-loss treatment, previous hair-restoration procedures, or inadequate clinical information were excluded. A structured clinical assessment documented demographic characteristics, onset and duration of shedding, distribution, associated symptoms, previous episodes, family history, systemic symptoms, medications, previous treatment, and potential precipitating factors, followed by scalp examination and hair-pull testing. Clinical history and examination were performed to identify endocrine, nutritional, medication-related, and systemic causes of diffuse hair shedding. Relevant medical history, precipitating factors, medications, and symptoms suggestive of systemic or endocrine disorders were assessed. Where clinically indicated, laboratory investigations included complete blood count, serum ferritin and iron status, thyroid-stimulating hormone, and selected biochemical or nutritional parameters to identify reversible causes and reduce misclassification. Trichoscopy was performed using a FotoFinder Medicam 1000 videodermoscope (FotoFinder Systems GmbH, Germany), with standardized images obtained from the frontal, temporal, and occipital scalp. Images were analysed using the TrichoLAB system. Hair density, follicular-unit density, hair-diameter diversity, shaft thickness, thin and thick hairs, single-, double- and triple-hair follicular units, vellus hairs, yellow dots, short and upright regrowing hairs, peripilar signs, empty follicular openings, and Sinclair scale were assessed using the predefined study criteria. Hair-diameter diversity was assessed as the proportion of hairs differing in diameter by $\geq 20\%$. Images with inadequate visualization were excluded from analysis. Trichoscopic assessment was performed by an experienced assessor who was aware of the clinical assessment, and no formal interobserver reliability assessment was performed. Participants were classified as having telogen effluvium (TE), female pattern hair loss (FPHL), or combined TE/FPHL based on predefined clinical and trichoscopic findings. Classification was performed using the overall clinical presentation and trichoscopic assessment, while acknowledging that features used for classification may overlap with those evaluated as diagnostic outcomes.

A 4-mm punch biopsy was obtained from a representative area of hair loss and processed for vertical and horizontal sectioning. Haematoxylin and eosin staining was used primarily to assess inflammatory infiltrates and general follicular architec-

ture, while immunohistochemistry was performed to characterize T-lymphocytic infiltration. Histopathological assessment focused primarily on the distribution and intensity of lymphocytic inflammatory infiltrates across predefined anatomical compartments; comprehensive quantitative assessment of terminal-to-vellus ratios, anagen-to-non-anagen ratios, miniaturization, and pigment casts was not performed. Immunohistochemistry was performed on paraffin-embedded sections using a CD3 rabbit monoclonal antibody (clone MRQ-39) to characterize T-lymphocytic infiltration; staining was interpreted by an experienced dermatopathologist blinded to the clinical classification, with CD3-positive cells assessed in the epidermal, papillary and reticular dermal, peri-infundibular, peribulbar, follicular-stelae, and subcutaneous compartments and graded as trace, mild, moderate, moderate-to-severe, or severe. Data was analysed using IBM SPSS Statistics version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean $\pm$ SD or median (IQR), as appropriate, and categorical variables as frequencies and percentages. Comparisons among the three diagnostic groups were performed using χ^2 or Fisher's exact tests for categorical variables and one-way ANOVA or Kruskal-Wallis tests for continuous variables, with appropriate post-hoc comparisons. Two-sided $p < 0.05$ was considered statistically significant. Ethical approval was obtained, and administrative authorization was obtained from Banadir Hospital. Written informed consent was obtained from all participants, and confidentiality and the principles of the Declaration of Helsinki were maintained throughout the study.

3. Results

During the study period, 95 women presenting with diffuse hair shedding were screened for eligibility. Of these, 86 met the predefined eligibility criteria, while 9 were excluded because of failure to meet the inclusion criteria or incomplete clinical information. Six women declined participation, and a total of 80 women completed the required clinical, trichoscopic, and histopathological assessments and were included in the final analysis.

3.1. Clinical Characteristics and Diagnostic Distribution

A total of 80 women with acute diffuse hair shedding were included in the analysis. The mean duration of hair shedding was 3.75 ± 1.0 months (range, 2 - 5 months). Onset was sudden in 46 participants (57.5%) and gradual in 34 (42.5%), while progression was mild in 64 (80.0%) and moderate in 16 (20.0%). The frontal scalp was the most frequently reported site of maximal visible hair loss (38 [47.5%]), followed by the frontoparietal region (34 [42.5%]) and generalized involvement (8 [10.0%]) (**Table 1**). Based on integrated clinical and trichoscopic assessment, 40 participants (50.0%) were classified as TE, 27 (33.8%) as FPHL, and 13 (16.3%) as combined TE/FPHL.

3.2. Trichoscopic Findings According to Diagnostic Group

Several trichoscopic features demonstrated significant differences among the

Table 1. Clinical characteristics of women with acute diffuse hair shedding.

Characteristic	Overall (N = 80)
Duration of hair shedding, months	
Mean $\pm$ SD	3.75 $\pm$ 1.0
Range	2 - 5
Onset, n (%)	
Gradual	34 (42.5)
Sudden	46 (57.5)
Progression, n (%)	
Mild	64 (80.0)
Moderate	16 (20.0)
Maximum site of obvious hair loss, n (%)	
Frontal	38 (47.5)
Frontoparietal	34 (42.5)
Generalized	8 (10.0)

SD, standard deviation.

three diagnostic groups (**Table 2**). Hair-diameter diversity $\geq 20\%$ was more frequent in FPHL (10/27 [37.0%]) and combined TE/FPHL (6/13 [46.2%]) than in TE (2/40 [5.0%]; $p = 0.019$). Similarly, single-hair follicular units were substantially more frequent in FPHL (63.0%) and combined TE/FPHL (46.2%) than in TE (5.0%; $p = 0.008$). Regional variation in hair-shaft diameter was also associated with diagnostic group, occurring in 61.5% of participants with combined TE/FPHL, 29.6% with FPHL, and 5.0% with TE ($p = 0.011$). Numerous frontal vellus hairs were observed in 37.0% of participants with FPHL and 46.2% with combined TE/FPHL but were absent in TE ($p = 0.009$). In contrast, short regrowing hairs were present in 75.0% of TE and 76.9% of combined TE/FPHL cases but were absent among participants with FPHL ($p = 0.004$). The peripilar sign was identified exclusively in FPHL (6/27 [22.2%]; $p = 0.021$), while empty frontal follicles were more frequent in FPHL (29.6%) than in TE (0%) or combined TE/FPHL (15.4%; $p = 0.031$). Multiple upright regrowing hairs were observed exclusively in the combined TE/FPHL group (4/13 [30.8%]; $p = 0.024$).

3.3. Quantitative Trichoscopic Parameters

Quantitative trichoscopic assessment showed significant differences in mean hair-shaft diameter and the proportion of thick hairs among the diagnostic groups. Mean hair-shaft diameter was expressed in micrometres (μm), while hair density was expressed as hairs/ cm^2 based on the standardized trichoscopic field analysed. Mean hair density and follicular-unit density did not differ significantly among TE, FPHL, and combined TE/FPHL. Mean hair-shaft diameter was significantly

Table 2. Trichoscopic features according to diagnostic group.

Trichoscopic feature	TE (n = 40)	FPHL (n = 27)	TE + FPHL (n = 13)	<i>p</i> value
Hair-diameter diversity $\geq 20\%$, n (%)	2 (5.0) ^a	10 (37.0) ^b	6 (46.2) ^b	0.019
Single-hair follicular units, n (%)	2 (5.0) ^a	17 (63.0) ^b	6 (46.2) ^b	0.008
Yellow dots, n (%)	2 (5.0) ^a	0.0000 ^a	0.0000 ^a	0.599
Hair-shaft diameter difference, n (%)	2 (5.0) ^a	8 (29.6) ^b	8 (61.5) ^c	0.011
Numerous frontal vellus hairs, n (%)	0.0000 ^a	10 (37.0) ^b	6 (46.2) ^b	0.009
Short regrowing hairs, n (%)	30 (75.0) ^a	0.0000 ^b	10 (76.9) ^a	0.004
Peripilar sign, n (%)	0.0000 ^a	6 (22.2) ^b	0.0000 ^a	0.021
Empty frontal follicles, n (%)	0.0000 ^a	8 (29.6) ^b	2 (15.4) ^{ab}	0.031
Multiple upright regrowing hairs, n (%)	0.0000 ^a	0.0000 ^a	4 (30.8) ^b	0.024
Mean hair density, hairs/cm ²	193.3 $\pm$ 25.0 ^a	192.7 $\pm$ 37.0 ^a	192.9 $\pm$ 33.0 ^a	0.999
Mean hair-shaft diameter, μ m	64.8 $\pm$ 8.0 ^a	58.3 $\pm$ 8.0 ^{ab}	55.6 $\pm$ 4.0 ^b	0.016
Thin hairs (<30 μ m), M $\pm$ SD	5.3 $\pm$ 2.6 ^a	8.3 $\pm$ 4.3 ^a	11.1 $\pm$ 5.1 ^a	0.181
Thick hairs (>50 μ m), M $\pm$ SD	74.4 $\pm$ 11.8 ^a	64.8 $\pm$ 16.5 ^{ab}	59.5 $\pm$ 9.2 ^b	0.041
Double follicular units, M $\pm$ SD	38.8 $\pm$ 9.1 ^a	36.4 $\pm$ 7.8 ^a	40.1 $\pm$ 5.6 ^a	0.578
Triple follicular units, M $\pm$ SD	37.3 $\pm$ 10.8 ^a	35.4 $\pm$ 10.1 ^a	31.6 $\pm$ 7.1 ^a	0.404
Total follicular units, M $\pm$ SD	85.4 $\pm$ 7.7 ^a	86.0 $\pm$ 11.2 ^a	90.9 $\pm$ 7.9 ^a	0.341
Derived Sinclair scale, M $\pm$ SD	2.1 $\pm$ 0.3 ^a	2.3 $\pm$ 0.4 ^a	2.3 $\pm$ 0.3 ^a	0.111

^a, ^b, ^c: Different superscript letters indicate statistically significant differences between groups; shared letters indicate no significant difference. $p < 0.05$ was considered statistically significant. TE, telogen effluvium; FPHL, female pattern hair loss; SD, standard deviation.

higher in FPHL and combined TE/FPHL than in TE ($p = 0.016$), while the proportion of thick hairs also differed significantly among groups ($p = 0.041$). No significant between-group differences were observed for overall hair density, thin-hair counts, double- or triple-hair follicular units, total follicular-unit density, or the derived Sinclair scale (all $p > 0.05$).

3.4. Histopathological Findings

Histopathological examination demonstrated predominantly mild lymphocytic infiltration involving the papillary and reticular dermis and peri-infundibular regions across the three diagnostic groups (Table 3). Peribulbar and follicular-stelae inflammatory infiltrates were generally absent or limited to trace/minimal levels. No differences were identified among TE, FPHL, and combined TE/FPHL in the distribution or intensity of lymphocytic infiltration within the papillary dermis, peri-infundibular region, reticular dermis, or peribulbar region. Follicular-stelae and subcutaneous inflammatory infiltrates were uniformly trace and therefore were not subjected to comparative statistical testing.

Table 3. Histopathological distribution of lymphocytic inflammatory infiltrates according to diagnostic group.

Histological compartment	TE	FPHL	TE + FPHL
Papillary dermis	Mild	Mild	Mild
Peri-infundibular region	Mild	Mild	Mild
Reticular dermis	Mild	Mild	Mild
Peribulbar region	Mild	Mild	Mild
Follicular stela	Trace	Trace	Trace
Subcutaneous tissue	Trace	Trace	Trace

TE, telogen effluvium; FPHL, female pattern hair loss.

3.5. CD3 Immunohistochemical Findings

CD3 immunohistochemistry confirmed the presence of T-lymphocytic infiltrates in multiple tissue compartments, including the epidermis, papillary and reticular dermis, peri-infundibular region, peribulbar region, follicular stela, and subcutaneous tissue. However, CD3-positive lymphocyte density did not differ significantly among the three diagnostic groups in any assessed compartment. The corresponding *p* values were 0.911 for epidermal, 0.725 for papillary dermal, 0.585 for peri-infundibular, 0.744 for reticular dermal, 0.769 for peribulbar, 0.365 for follicular-stela, and 0.129 for subcutaneous infiltration (**Table 4**).

Table 4. CD3-positive lymphocytic infiltration according to tissue compartment.

Tissue compartment	<i>p</i> value
Epidermis	0.911
Papillary dermis	0.725
Peri-infundibular region	0.585
Reticular dermis	0.744
Peribulbar region	0.769
Follicular stela	0.365
Subcutaneous tissue	0.129

3.6. Alopecia Areata Incognita

Although AAI was included in the initial differential diagnosis because of its clinical overlap with acute diffuse hair shedding, none of the 80 participants fulfilled the predefined clinical, trichoscopic, and histopathological criteria for AAI. Accordingly, AAI was not included as a comparative diagnostic group.

4. Discussion

The present study demonstrates that acute diffuse hair shedding in women represents a diagnostically heterogeneous clinical presentation rather than a uniform

manifestation of telogen effluvium (TE). Among the 80 women evaluated, TE accounted for 50.0% of cases, female pattern hair loss (FPHL) for 33.8%, and combined TE/FPHL for 16.3%. This distribution is consistent with the diagnostic challenge outlined in the Background, where acute diffuse shedding was emphasized as a phenotype shared by biologically distinct disorders [2] [4] [5]. The finding that half of the participants had FPHL either alone or in combination with TE is particularly important because pattern hair loss may initially present with increased shedding before clinically obvious follicular miniaturization becomes apparent [4] [6]. Thus, the clinical label of “acute diffuse hair shedding” should not automatically be equated with isolated TE.

The temporal characteristics of the cohort were broadly compatible with acute TE. The mean duration of shedding was 3.75 ± 1.0 months, and 57.5% of participants reported sudden onset. TE is characterized by disruption of the normal hair cycle, with premature transition of follicles toward catagen and telogen and subsequent shedding of telogen hairs [2] [3]. However, the present findings also demonstrate why symptom duration and onset alone are insufficient for establishing the underlying diagnosis. Although acute onset is compatible with TE, one-third of participants had FPHL and a further 16.3% had combined TE/FPHL. This supports the concept that TE may coexist with an underlying chronic miniaturization disorder and that the distinction between an acute shedding episode and the background follicular phenotype requires additional assessment [2] [5].

The distribution of visible hair loss provides further evidence of this diagnostic complexity. Frontal loss was observed in 47.5% of participants and frontoparietal loss in 42.5%, whereas only 10.0% had generalized visible reduction. Frontal and frontoparietal involvement is compatible with the clinical distribution of FPHL, in which progressive follicular miniaturization can produce widening of the central part and reduced density over the frontal and mid-scalp regions [4] [6]. Nevertheless, distribution alone cannot reliably distinguish FPHL from TE in patients with diffuse shedding, reinforcing the rationale for the multimodal diagnostic strategy proposed in the Background.

Trichoscopy provided the clearest evidence of phenotypic differences among the diagnostic groups. Several trichoscopic variables traditionally associated with follicular miniaturization were significantly more frequent in FPHL and combined TE/FPHL than in isolated TE. Hair-diameter diversity of $\geq 20\%$ was present in 37.0% of women with FPHL and 46.2% with combined TE/FPHL, compared with only 5.0% with TE ($p = 0.019$). Similarly, single-hair follicular units occurred in 63.0% of FPHL and 46.2% of combined cases, compared with 5.0% of TE ($p = 0.008$). These findings closely correspond to the trichoscopic principles described in the Background, particularly the importance of hair-diameter variability and changes in follicular-unit composition as indicators of follicular miniaturization [7] [8]. Although the observed trichoscopic features were useful in characterizing the structural differences between TE, FPHL, and combined TE/FPHL, these findings should be interpreted cautiously because some features may have contributed

to the initial diagnostic classification. Therefore, the observed associations represent descriptive differences between the predefined clinical groups rather than independently validated diagnostic discriminators. Further studies using independent diagnostic criteria and blinded assessment are required to establish the diagnostic performance and generalizability of these trichoscopic features.

The significant difference in hair-shaft diameter variability provides further support for this interpretation. A difference in shaft diameter was observed in 29.6% of FPHL cases and 61.5% of combined TE/FPHL cases, compared with only 5.0% of TE ($p = 0.011$). Hair-diameter variability is a recognized consequence of asynchronous follicular miniaturization and is therefore more characteristic of pattern hair loss than of isolated TE [7] [8]. The particularly high frequency in the combined group is biologically plausible because these patients may simultaneously demonstrate an acute telogen shedding process and pre-existing follicular miniaturization. The finding also supports the observation in the Background that TE and pattern hair loss frequently overlap, making their distinction clinically important [2] [5].

Numerous frontal vellus hairs were identified in 37.0% of FPHL and 46.2% of combined TE/FPHL cases but were absent in TE ($p = 0.009$). The presence of increased vellus hairs in the frontal scalp is consistent with progressive miniaturization and provides an additional trichoscopic marker supporting FPHL [7] [8]. Similarly, the peripilar sign was identified in 22.2% of women with FPHL and was absent in both TE and combined TE/FPHL ($p = 0.021$). Peripilar signs have been described in association with androgenetic/pattern hair loss and may reflect perifollicular inflammatory or fibrotic changes accompanying miniaturization [7] [8] [17]. The absence of this feature in the combined group should, however, be interpreted cautiously because trichoscopic signs are not universally present in every patient with pattern hair loss.

Empty frontal follicles were also significantly associated with the diagnostic groups, occurring in 29.6% of FPHL, 15.4% of combined TE/FPHL and none of the TE cases ($p = 0.031$). Empty follicular openings may occur in several non-scarring alopecias and therefore should not be interpreted in isolation [8] [9]. In the present study, however, their concentration in FPHL and combined cases, together with hair-diameter diversity, vellus hairs and single-hair follicular units, strengthens their interpretation as part of a broader miniaturization phenotype rather than as an independent diagnostic marker.

In contrast, short regrowing hairs were observed in 75.0% of TE and 76.9% of combined TE/FPHL cases but were absent in FPHL ($p = 0.004$). This finding is consistent with the expected biology of TE, in which follicles recovering from a shedding episode re-enter anagen and produce short regrowing hairs [2] [3] [9]. Their high frequency in the combined group suggests that active telogen shedding and subsequent regrowth may remain evident even when underlying pattern hair loss is present. Consequently, the coexistence of miniaturization markers and regrowing hairs may be particularly useful for recognizing TE superimposed on

FPHL.

Multiple upright regrowing hairs were observed exclusively in the combined group (30.8%; $p = 0.024$). Although this observation may reflect active follicular recovery in patients experiencing both acute shedding and underlying miniaturization, the small number of participants in the combined group requires caution in interpreting this finding. It should therefore be regarded as a potentially supportive rather than a defining feature of combined TE/FPHL until confirmed in larger cohorts.

Interestingly, yellow dots were uncommon and did not differ significantly between groups ($p = 0.599$). Yellow dots are classically associated with alopecia areata but can occur in other hair disorders and are therefore not entirely disease-specific [8] [9]. Their low frequency in the present cohort is compatible with the subsequent finding that no participant fulfilled the predefined criteria for alopecia areata incognita (AAI).

The quantitative trichoscopic findings provide additional support for follicular miniaturization as the major distinguishing feature between TE and FPHL. Mean hair density did not differ significantly between the groups (193.3 ± 25.0 hairs/cm² in TE, 192.7 ± 37.0 hairs/cm² in FPHL and 192.9 ± 33.0 hairs/cm² in combined TE/FPHL; $p = 0.999$). This finding is important because it illustrates a limitation of relying solely on total hair density. Early pattern hair loss may involve qualitative changes in shaft caliber and follicular-unit composition before a substantial reduction in overall hair count becomes measurable [7] [8]. Thus, apparently preserved total density should not exclude early FPHL.

In contrast, mean hair-shaft diameter differed significantly across groups, decreasing from 64.8 ± 8.0 μ m in TE to 58.3 ± 8.0 μ m in FPHL and 55.6 ± 4.0 μ m in combined TE/FPHL ($p = 0.016$). Similarly, the proportion of thick hairs > 50 μ m was significantly lower in FPHL and combined TE/FPHL than in TE ($p = 0.041$). These findings provide objective quantitative evidence of follicular miniaturization and complement the qualitative trichoscopic findings of hair-diameter diversity, vellus hairs and altered follicular units [7] [8]. The convergence between qualitative and quantitative parameters strengthens the interpretation that reduced shaft caliber, rather than absolute hair density, is a major discriminator of pattern hair loss in this cohort.

The mean Sinclair scale did not differ significantly among groups ($p = 0.111$), despite significant differences in several objective trichoscopic parameters. This apparent discrepancy is clinically informative. The Sinclair scale is a global clinical grading system, whereas trichoscopy permits direct assessment of individual follicular and hair-shaft characteristics. Consequently, subtle early miniaturization may be detectable trichoscopically before producing a sufficiently marked change in global clinical grading. This observation supports the position established in the Background that trichoscopy can reveal abnormalities that are not readily apparent on conventional examination [7].

Histopathology demonstrated predominantly mild lymphocytic infiltrates within

the papillary and reticular dermis and peri-infundibular regions across all three diagnostic groups, with no statistically significant differences between TE, FPHL and combined TE/FPHL. Peribulbar and follicular-stelae inflammation was limited or minimal. These findings are consistent with the limitations of histopathology in non-scarring diffuse alopecias described in the Background [10] [11]. In particular, TE may show relatively subtle or nonspecific histological abnormalities, while mild inflammatory infiltrates may not necessarily indicate a specific inflammatory alopecia.

The absence of significant differences in inflammatory distribution should therefore not be interpreted as evidence that histopathology is unhelpful. Rather, it demonstrates that conventional inflammatory features alone may have limited discriminatory capacity when the principal differential diagnoses are TE and FPHL. Histopathological diagnosis of diffuse alopecia requires appropriate sampling, orientation and integration of architectural findings with the clinical and trichoscopic phenotype [10] [11]. Quantitative follicular parameters, including terminal-to-vellus ratios and anagen-to-non-anagen relationships, may provide greater discriminatory information than nonspecific inflammatory infiltrates alone [11].

The relatively limited follicular inflammatory response in the present cohort is also relevant to the absence of AAI. Diffuse forms of alopecia areata can sometimes demonstrate less conspicuous peribulbar inflammation than classic patchy disease, making the diagnosis challenging on routine histology [12] [13]. Nevertheless, the absence of the characteristic clinical, trichoscopic and histopathological constellation required by the study protocol argues against AAI as a major contributor to the present cohort.

CD3 immunohistochemistry demonstrated T-lymphocytic infiltration in multiple tissue compartments, including the epidermis, papillary and reticular dermis, peri-infundibular and peribulbar regions, follicular stelae and subcutaneous tissue. However, none of these distributions differed significantly between the three diagnostic groups. These findings are important in the context of the Background, which identified IHC as a potentially useful adjunct for characterizing immune-mediated follicular injury [12]-[16].

The absence of significant between-group differences indicates that the mere presence of CD3-positive lymphocytes is insufficient to distinguish TE from FPHL in this cohort. This does not negate the biological relevance of T lymphocytes in alopecia areata; rather, it suggests that CD3-positive cells may be present at low or variable levels in different forms of diffuse hair loss and that their diagnostic value depends on their density, distribution and anatomical localization. Previous work has suggested that the localization of CD3-positive lymphocytes within follicular fibrous stelae may be particularly useful in distinguishing diffuse alopecia areata from pattern hair loss, rather than simply documenting T-cell presence throughout the dermis [18]. Thus, future studies may benefit from more standardized quantitative scoring and compartment-specific analysis of CD3-pos-

itive cells.

The present IHC findings also reinforce the complementary nature of the diagnostic modalities. Histopathology and IHC did not provide the same degree of group separation as the trichoscopic parameters. This does not establish that IHC lacks diagnostic value in diffuse alopecia; rather, in this particular cohort, CD3 staining did not significantly discriminate among TE, FPHL and combined TE/FPHL.

No participant fulfilled the predefined criteria for AAI. This is noteworthy because AAI represents an important clinical mimic of acute diffuse shedding and may occur without the sharply demarcated patches characteristic of conventional alopecia areata [12] [13]. AAI has been described as a diffuse form of alopecia areata characterized by acute or subacute shedding, numerous short regrowing hairs and other trichoscopic abnormalities, with histopathological evidence of increased telogen structures and variable peribulbar inflammation [19] [20]. The absence of AAI in the present cohort therefore does not imply that the entity is irrelevant to patients with acute diffuse shedding; instead, it highlights the importance of actively excluding it when clinical and trichoscopic findings are atypical for TE.

Taken together, the findings support the multimodal diagnostic framework proposed in the Background. Clinical assessment established the temporal characteristics and distribution of shedding but could not by itself adequately explain the heterogeneity of the cohort. Trichoscopy provided the most evident phenotypic discrimination, particularly through hair-diameter diversity, single-hair follicular units, frontal vellus hairs, shaft-diameter differences, peripilar signs and empty follicles. Quantitative trichoscopy further demonstrated differences in shaft caliber despite comparable total hair density. Histopathology provided tissue-level assessment but predominantly demonstrated mild and nonspecific inflammatory changes, while CD3 IHC demonstrated T-cell presence without significant group-level discrimination.

These findings therefore support the concept that the diagnostic modalities are complementary rather than interchangeable [4] [7] [10]. The clinical examination identifies the syndrome; trichoscopy characterizes the follicular phenotype; histopathology assesses follicular architecture and tissue inflammation; and IHC can provide additional immunophenotypic information when an immune-mediated process is suspected. Importantly, the present data suggest that the greatest diagnostic yield in women with acute diffuse shedding may arise from the integration of clinical assessment with systematic trichoscopic evaluation, reserving biopsy and IHC for diagnostically uncertain or atypical presentations.

5. Conclusion

Acute diffuse hair shedding in women represents a heterogeneous diagnostic presentation, with telogen effluvium, female pattern hair loss, and their coexistence accounting for the majority of cases in this cohort. Trichoscopy provided the

most informative discrimination between diagnostic groups, particularly through assessment of hair-diameter variability, follicular-unit composition, vellus hairs, regrowing hairs, and quantitative shaft caliber. Histopathology demonstrated predominantly mild and nonspecific lymphocytic inflammation, while CD3 immunohistochemistry did not significantly differentiate the diagnostic groups. The findings support an integrated clinical and trichoscopic approach to acute diffuse hair shedding, with histopathology and immunohistochemistry serving as complementary investigations in diagnostically uncertain or atypical cases.

Study Limitations

This study has limitations that generality of the results should be done with some caution. First, its cross-sectional design and relatively small sample size, particularly the 13 participants in the combined TE/FPHL group, limit statistical power and preclude assessment of diagnostic evolution or causal relationships. Second, the study was conducted at a single tertiary hospital in Mogadishu, which may limit the generalizability of the findings to other Somali or African populations. The consecutive recruitment approach may have reduced selection bias by allowing eligible women presenting during the study period to be assessed systematically. Nevertheless, the hospital-based nature of the study may have introduced selection bias, as women who presented to Banadir Hospital may not represent all women with acute diffuse hair shedding in the wider community. Therefore, although the analyzed cohort comprised women who met the eligibility criteria and completed the study procedures during the recruitment period, the findings may not be fully representative of all eligible patients in the broader population. Third, diagnostic classification was based primarily on integrated clinical and trichoscopic assessment, and the absence of a universally accepted reference standard for acute diffuse hair shedding may introduce classification uncertainty. Fourth, although histopathology and CD3 immunohistochemistry were performed, quantitative follicular parameters such as terminal-to-vellus ratios and detailed quantitative CD3-cell counts were not comprehensively incorporated into the comparative analysis. Finally, potential variation in hair characteristics, grooming practices, environmental exposures, nutritional factors, and unmeasured systemic or hormonal influences may have affected the observed trichoscopic and clinicopathological findings. Larger, multicentre prospective studies incorporating standardized quantitative histopathology, immunohistochemical scoring, laboratory evaluation, and longitudinal follow-up are warranted to validate these findings and develop more robust diagnostic algorithms for acute diffuse hair shedding.

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Author Contributions

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Conflicts of Interest

The authors declare no conflicts of interest.

References

- [1] Rebora, A. (2019) Telogen Effluvium: A Comprehensive Review. *Clinical, Cosmetic and Investigational Dermatology*, **12**, 583-590. <https://doi.org/10.2147/ccid.s200471>
- [2] Asghar, F., Shamim, N., Farooque, U., Sheikh, H. and Aqeel, R. (2020) Telogen Effluvium: A Review of the Literature. *Cureus*, **12**, e8320. <https://doi.org/10.7759/cureus.8320>
- [3] Jimeno Ortega, I. and Stefanato, C.M. (2023) Telogen Effluvium: A 360 Degree Review. *Italian Journal of Dermatology and Venereology*, **158**, 457-466. <https://doi.org/10.23736/s2784-8671.23.07579-5>
- [4] Werner, B. and Mulinari-Brenner, F. (2012) Clinical and Histological Challenge in the Differential Diagnosis of Diffuse Alopecia: Female Androgenetic Alopecia, Telogen Effluvium and Alopecia Areata—Part I. *Anais Brasileiros de Dermatologia*, **87**, 742-747. <https://doi.org/10.1590/s0365-05962012000500012>
- [5] Torres, F. (2015) Female Pattern Alopecia and Telogen Effluvium: Figuring out Diffuse Alopecia. *Seminars in Cutaneous Medicine and Surgery*, **34**, 67-71. <https://doi.org/10.12788/j.sder.2015.0142>
- [6] Vujovic, A. and Del Marmol, V. (2014) The Female Pattern Hair Loss: Review of Etiopathogenesis and Diagnosis. *BioMed Research International*, **2014**, Article ID: 767628. <https://doi.org/10.1155/2014/767628>
- [7] Inui, S. (2010) Trichoscopy for Common Hair Loss Diseases: Algorithmic Method for Diagnosis. *The Journal of Dermatology*, **38**, 71-75. <https://doi.org/10.1111/j.1346-8138.2010.01119.x>
- [8] Saqib, N.U., Bhat, Y.J., Shah, I.H., Haq, I., Devi, R., Shah, A.A., *et al.* (2021) Assessment, Reliability, and Validity of Trichoscopy in the Evaluation of Alopecia in Women. *International Journal of Women's Dermatology*, **7**, 458-465. <https://doi.org/10.1016/j.ijwd.2021.02.002>
- [9] Rakowska, A., Slowinska, M., Kowalska-Oledzka, E., Olszewska, M. and Rudnicka, L. (2009) Dermoscopy in Female Androgenic Alopecia: Method Standardization and Diagnostic Criteria. *International Journal of Trichology*, **1**, 123-130. <https://doi.org/10.4103/0974-7753.58555>
- [10] Stefanato, C.M. (2010) Histopathology of Alopecia: A Clinicopathological Approach to Diagnosis. *Histopathology*, **56**, 24-38. <https://doi.org/10.1111/j.1365-2559.2009.03439.x>
- [11] Ieremia, E. and Stefanato, C.M. (2023) The Role of Hair Follicle Counts and Ratios

- in the Histopathological Assessment of Androgenic Alopecia, Alopecia Areata and Telogen Effluvium: Does Counting “Count”? *Human Pathology*, **140**, 233-239. <https://doi.org/10.1016/j.humpath.2023.03.015>
- [12] Darwin, E., Hirt, P.A., Fertig, R., Doliner, B., Delcanto, G. and Jimenez, J. (2018) Alopecia Areata: Review of Epidemiology, Clinical Features, Pathogenesis, and New Treatment Options. *International Journal of Trichology*, **10**, 51-60. https://doi.org/10.4103/ijtr.ijtr_99_17
- [13] Rajabi, F., Drake, L.A., Senna, M.M. and Rezaei, N. (2018) Alopecia Areata: A Review of Disease Pathogenesis. *British Journal of Dermatology*, **179**, 1033-1048. <https://doi.org/10.1111/bjd.16808>
- [14] Kamyab, K., Rezvani, M., Seirafi, H., Mortazavi, S., Teymourpour, A., Abtahi, S., *et al.* (2019) Distinguishing Immunohistochemical Features of Alopecia Areata from Androgenic Alopecia. *Journal of Cosmetic Dermatology*, **18**, 422-426. <https://doi.org/10.1111/jocd.12677>
- [15] Herskovitz, I., de Sousa, I.C. and Tosti, A. (2013) Vellus Hairs in the Frontal Scalp in Early Female Pattern Hair Loss. *International Journal of Trichology*, **5**, 118-120. <https://doi.org/10.4103/0974-7753.125601>
- [16] Bhamla, S.A., Dhurat, R.S. and Saraogi, P.P. (2013) Is Trichoscopy a Reliable Tool to Diagnose Early Female Pattern Hair Loss? *International Journal of Trichology*, **5**, 121-125. <https://doi.org/10.4103/0974-7753.125603>
- [17] Deloche, C., de Lacharrière, O., Misciali, C., Piraccini, B.M., Vincenzi, C., Bastien, P., *et al.* (2004) Histological Features of Peripilar Signs Associated with Androgenetic Alopecia. *Archives of Dermatological Research*, **295**, 422-428. <https://doi.org/10.1007/s00403-003-0447-y>
- [18] Kolivras, A. and Thompson, C. (2016) Distinguishing Diffuse Alopecia Areata (AA) from Pattern Hair Loss (PHL) Using CD3⁺ T Cells. *Journal of the American Academy of Dermatology*, **74**, 937-944. <https://doi.org/10.1016/j.jaad.2015.12.011>
- [19] Alessandrini, A., Starace, M., Bruni, F., Brandi, N., Baraldi, C., Misciali, C., *et al.* (2019) Alopecia Areata Incognita and Diffuse Alopecia Areata: Clinical, Trichoscopic, Histopathological, and Therapeutic Features of a 5-Year Study. *Dermatology Practical & Conceptual*, **9**, 272-277. <https://doi.org/10.5826/dpc.0904a05>
- [20] Miteva, M., Misciali, C., Fanti, P.A. and Tosti, A. (2012) Histopathologic Features of Alopecia Areata Incognito: A Review of 46 Cases. *Journal of Cutaneous Pathology*, **39**, 596-602. <https://doi.org/10.1111/j.1600-0560.2012.01896.x>