The “Fringe Sign” A Useful Clinical Finding in Traction Alopecia of the Marginal Hair Line

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Abstract

Background: Traction alopecia is hair loss due to prolonged or repetitive tension on the hair. Diagnostic challenges may be encountered if the clinical suspicion for traction is not high, or if the history of traction is remote or not obtained. Since pathologic features can vary dramatically with the stage of the disorder clinico-pathologic correlation is essential. We have made the observation that the presence of retained hairs along the frontal or temporal rim, which we termed the “fringe sign”, is a finding that can be seen in both early and late traction alopecia, and thus may be a useful clinical marker of the condition.

Objective: To determine the frequency of the fringe sign in a series of patients with a diagnosis of traction alopecia.

Methods: This was a retrospective single-center review.

Results: Over a 3.5 year period the diagnosis of traction alopecia was made in 41 women. Twelve of the 41 patients were Hispanic (29%). The average age of our cohort was 34. Thirty-five (85%) of all women and 100% of women who had traction involving the marginal hair line had the fringe sign. The majority of African American women (54%) compared to 17% of the Hispanic women had some clinical sign of scalp inflammation (most frequently scalp scaling). Fourteen biopsies (58%) were available for review. Histopathologic findings included retained sebaceous glands (100%) and an increase in veilus-sized hairs (50%), a decrease in terminal hairs (100%), fibrotic fibrous tracts (100%), and sparse lymphocytic inflammation (57%). Trichomalacia was only noted in only one of the biopsies.

Limitations: Retrospective analysis, uncontrolled study.

Conclusions: Hispanic women as well as African American women are at high risk for traction alopecia. The fringe sign was a sensitive and specific clinical feature of traction alopecia when it involved the marginal hair line. Retained sebaceous glands, decreased terminal hairs, and fibrotic fibrous tracts were noted in all histopathologic specimens.

Keywords: Traction alopecia; Hispanic; Hairstyles; Histopathology; African American

Abbreviations: TA: Traction Alopecia; PPAR: Peroxisome Proliferator Activated Receptor

Introduction

Traction alopecia (TA) is a term used to describe hair loss which occurs due to prolonged or repetitive tension on the hair. TA was first described in 1907 in subjects from Greenland who developed hair loss along the hairline due to the prolonged wearing of tight ponytails [1]. Subsequently most of the literature has focused on the prevalence of TA in people of African descent [2,3]. However, TA affects people of different ethnic backgrounds and is the result of an individual’s hair care practices and can have a large variation in its pattern of clinical presentation. Diagnostic challenges may be encountered if the clinical suspicion for traction is not high, or if the history of traction is remote or not obtained. Since pathologic features are biphasic, and show varying features in early and late stage disease, appropriate clinico-pathologic correlation is essential in securing a diagnosis of TA. In patients who do not give a clear history of tight hairstyles, the clinical differential is broad and can include alopecia areata, androgeneic alopecia, telogen effluvium, trichotillomania, and primary lymphocytic cicatricial alopecias (lichen planopilaris, central centrifugal cicatricial alopecia, pseudopelade of Brocq, and frontal fibrosing alopecia). TA is most commonly misdiagnosed as alopecia areata as these two disorders can share many common clinical features such as a patchy or band-like pattern of hair loss [14]. Thus identification of sensitive and specific clinical markers of TA would be a useful aide to clinicians and pathologists in distinguishing TA from other conditions.

Clinically, TA most often affects the frontal and temporal scalp. However, TA has been extensively reported in the literature to occur on many different regions of the scalp. The location of TA is dependent on an individual’s hair care practices which may or may not be related to his/her racial or ethnic background. For example, frontal and parietal alopecia has been described in the literature as a result of twisting their uncut hair tightly on the scalp [5]. Submandibular TA has also been reported in a Sikh male who tied his beard in a tight knot below the chin [6]. Young and adult African-American females, who develop TA from braids or hair weaves, have hair loss localized to the temporal scalp as well as anterior and superior to the ears. Females who frequently wear their hair in a tight chignon or bun can develop hair loss confined to...
the occipital or temporal scalp. This pattern of TA has been described in European as well as Japanese women and in ballerinas [7,8]. In Sudanese women tight braiding causes hair loss on the vertex of the scalp and young Zande develop TA at the frontal scalp secondary to wooden combs placed horizontally on the scalp [9]. TA in Amish American women is noted on the temporal scalp where the religious head dressing is pinned (personal observation PM). In addition, recent case series have reported traction alopecia from hair extensions [10].

It is evident that TA occurs in people of different ethnic backgrounds and is the result of an individual's hair-styling and hair care practices. It is important to note that studies in African females have shown that the likelihood of developing TA increases when traction is applied to chemically processed hair [2,11]. The frequency of hair relaxing, however, does not appear to affect susceptibility of developing TA [11]. Chemically processed hair may be less resistant to TA than natural hair. Patients who develop any symptoms with hair-dressing (including pain, pimples, stinging, or crusts) have also been shown to be at increased risk of developing TA [11]. The likelihood of developing TA also increases with age, which is likely the result of a longer history of these hair practices.

We have made the observation that the presence of retained hairs along the frontal and/or temporal rim, which we termed the "fringe sign", is a common finding in patients with traction alopecia. In this study we sought to determine how frequently the fringe sign was noted in a series of patients with a diagnosis of traction alopecia. Also, we aimed to review the frequency of other clinical signs and histologic markers reported for these patients.

Methods

Over a 3.5 year period in a specialty hair referral clinic (Kaiser Permanente Vallejo- Northern California), the diagnosis of TA was made in 41 women. The diagnosis of TA was made based on a clinical finding of patchy non-scarring alopecia in the setting of tight hairstyles. When the clinical history of tight hairstyles was remote or not obtained, the diagnosis of TA was confirmed by scalp biopsy. A retrospective chart review was undertaken. Photographs and histologic slides were also reviewed when available. Data was collected on whether the following clinical signs of traction were noted in the chart: the fringe sign, scalp signs of inflammation (scale, pustules, erythema, papules), and the presence of follicular markings. Histologic findings that were reviewed included: retained sebaceous glands, trichomalacia, increased catagen and telogen hairs, number of terminal hairs, vellus-sized hairs (0.03mm), fibrotic fibrous tracts, and the presence of inflammation.

Results

Although the majority of the women were African-American, 24 (58.5%), 12 (29.2%) were of Hispanic ancestry. A summary of the demographics and clinical findings are presented (Table 1 and Table 2). The ages of the patients ranged from 15-66 years with an average age at presentation of 34 years. Ninety percent of women reported a duration of hair loss of one year or greater. All of the Hispanic women shared a history of having long thick hair and wearing their hair back in a tight ponytail for many years prior to noticing hair loss (Table 2). The majority of the women reported tight hairstyles starting in their teens or childhood.

Alopecia was seen in the distribution of frontal/temporal and less commonly the vertex and occipital scalp; the extent of alopecia varied from mild to extensive. The fringe sign was a sensitive and specific clinical finding in both mild early and extensive late stage traction alopecia when it involved the marginal scalp (Figures 1-4). Six African American women did not have the fringe sign on exam, but instead had patchy alopecia due to use of glued-in or tightly sewn-in weft-extensions that were placed at the vertex or occipital scalp. There was a decreased density of follicular markings in all of the patients. The majority of African American women (54%) compared to 17% of the Hispanic women had some clinical sign of scalp inflammation (most frequent finding was scalp scaling). Two of the women (5%) reported a positive history of androgenetic alopecia and showed miniaturization on exam. Almost one third of the African American women with traction alopecia also had an additional diagnosis of central centrifugal cicatricial alopecia. In 2 patients an alternative diagnosis of alopecia areata had been made clinically. One patient was also given an alternative diagnosis of primary lymphocytic cicatricial alopecia based on histopathology [12-14]. However review of the histopathology and correlation with the clinical findings and history confirmed the diagnosis of TA in this subject.

Fourteen biopsies (58%) were available for review. Histopathologic

<table>
<thead>
<tr>
<th>Category</th>
<th>Total no. of patients</th>
<th>No. (%)</th>
<th>Average (range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>African-American</td>
<td>24</td>
<td>(58.5)</td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>12</td>
<td>(29.2)</td>
<td></td>
</tr>
<tr>
<td>Other*</td>
<td>2</td>
<td>(4.9)</td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>1</td>
<td>(2.4)</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
<td>(4.9)</td>
<td></td>
</tr>
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</table>

*Other denotes multiracial

Table 1: Patient demographics.

<table>
<thead>
<tr>
<th>Clinical findings</th>
<th>African-American N(%)</th>
<th>Hispanic N(%)</th>
<th>Other N(%)</th>
<th>Caucasian N(%)</th>
<th>Unknown N(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fringe sign</td>
<td>18 (75)</td>
<td>12 (100)</td>
<td>2 (100)</td>
<td>1 (100)</td>
<td>2 (100)</td>
</tr>
<tr>
<td>Concurrent diagnosis</td>
<td>Central centrifugal cicatrical alopecia</td>
<td>7 (29)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Trichorrhexis nodosa</td>
<td>5 (21)</td>
<td>1 (8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Androgenetic or senescent alopecia</td>
<td>2 (17)</td>
<td></td>
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</table>

Table 2: Clinical findings.

<table>
<thead>
<tr>
<th>Category</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retained sebaceous glands</td>
<td>14 (100)</td>
</tr>
<tr>
<td>Increased vellus-sized hairs</td>
<td>12 (50)</td>
</tr>
<tr>
<td>Decreased terminal hairs</td>
<td>14 (100)</td>
</tr>
<tr>
<td>Fibrotic fibrous tracts</td>
<td>14 (100)</td>
</tr>
<tr>
<td>Trichomalacia</td>
<td>1 (7)</td>
</tr>
</tbody>
</table>

Table 3: Histologic findings in 14 patients.
findings included retained sebaceous glands (100%) a decrease in the ratio of terminal/ vellus-sized hairs (50%), a decrease in terminal hairs (100%), fibrotic fibrous tracts (100%) (Figure 5), and sparse lymphocytic inflammation (57%). Trichomalacia was only noted in only one of the biopsies (Table 3).

Discussion

History and exam

The relatively high prevalence of Hispanic women with TA in this series may represent the geographic demographics of the referral clinic. However, a heightened awareness of the risk of TA in all Hispanic women who wear tight ponytails with long hair will likely improve early diagnosis of the disorder, when hair loss is still reversible. Although diagnosis of TA is usually highly suggestive based on clinical history and presentation, the diagnosis was not readily made in our Hispanic patients. Some gave a clear history of chronic tight ponytails and had insight into the likely cause of hair loss. Others however, reported “sudden onset” hair loss and only upon specific questioning of hair care practices admitted to a history of chronic traction. It should be noted that each individual has a different tolerance to the amount of tension/traction on the hair follicle required to produce symptoms of pain or signs of TA. For instance, a patient may complain that the hairstyle is too tight and the scalp aches but will never develop TA while another patient may not experience pain but will still develop TA. Since the fringe sign was noted on exam in all of our patients with marginal alopecia, and in some cases was the trigger for further questioning and heightened clinical suspicion for TA we propose that it can be used as a characteristic clinical finding that can alert the clinician to the diagnosis of TA. When combined with histopathologic examination, an accurate diagnosis of TA can be made.

Histopathology

Early, the histopathology of TA shows trichomalacia, increased numbers of telogen and catagen hairs, a normal number of terminal follicles, and preserved sebaceous glands. At some point there may be “follicular drop-out” of the terminal hairs where the follicles seem to have disappeared but the vellus-sized hairs are intact [12]. With longstanding TA, sebaceous glands are present but may be decreased and vellus-sized hairs may be seen (Figure 6 A-B). There is a decrease in the number of terminal follicles, which are replaced with fibrotic fibrous tracts (Figure 6 C). Inflammation is little to absent in longstanding TA but may be mild in some cases of early TA. In our 14 patients with biopsy reports (Table 3) histopathology revealed findings consistent with the diagnosis of long-standing TA (Figure 4). Indeed, only one case showed evidence of trichomalacia, a typical finding of earlier stage TA. The fact that the majority of patients in the cohort (90%) had a history of hair loss longer than one year is in keeping with the report...
Biopsy from a patient with traction alopecia reveals a slightly reduced follicular density, follicular miniaturization, and retained sebaceous glands (hematoxylin-eosin, original magnification x 40). (B) Follicular miniaturization and retained sebaceous glands (hematoxylin-eosin, original magnification x 100). (C) Many fibrotic fibrous tracts (lower half) were present (hematoxylin-eosin, original magnification x 200).

Figure 6: (A) Biopsy from a patient with traction alopecia reveals a slightly reduced follicular density, follicular miniaturization, and retained sebaceous glands (hematoxylin-eosin, original magnification x 200).

Clinico-pathologic correlation and differential diagnosis

Clinico-pathologic correlation is key and essential for both the clinician and the histopathologist since the histopathology of TA varies with duration and has some overlapping features with other disorders commonly considered in the differential diagnosis of TA. The earliest clinical sign of traction on the scalp is perifollicular erythema, which may progress to folliculitis with continued traction. Scattered broken terminal hairs are often seen within areas of hair loss. In some cases 3-7mm fine yellow-white keratin cylinders that encircle the hair shaft may form [16]. These are termed peri-pilar casts. The majority of African American women (54%) in the cohort compared to 17% of the Hispanic women had some clinical sign of scalp inflammation (most frequent finding was scalp scaling). Two of the Hispanic patients in our series were noted to have perifollicular erythema and complained of scalp discomfort. However, the majority of women had no evidence of mostly late stage findings on biopsy specimens. An additional factor may have been a bias in the clinic to biopsy patients with late stage disease, or a remote history of traction more frequently.

Histologically, in active alopecia areata peribulbar lymphocytic inflammation and absence of fibrotic fibrous tracts distinguishes this disease from TA. The histopathologic findings of androgenetic alopecia and longstanding TA can be similar in that both can show follicular miniaturization, retained sebaceous lobules, and fibrotic fibrous tracts. However in androgenetic alopecia follicular miniaturization is more prominent and the number of terminal follicles is greater than in longstanding TA.

In both primary lymphocytic cicatricial alopecias (PCAs) and longstanding TA, the number of terminal follicles is decreased. Both can show fibrotic fibrous tracts, but perifollicular fibrosis and inflammation are characteristic of the PCAs and not seen in TA. In PCAs the loss of sebaceous glands is an early finding, whereas in TA, both early and late, sebaceous glands are retained [12]. This significant difference in sebaceous gland pathology highlights the likely differences in pathogenesis of these two disorders. It has recently been determined that in lichen planopilaris the underlying pathology occurs due to loss of activity of the peroxisome proliferator activated receptor gamma (PPAR-gamma) in the sebaceous gland[14].

In a recent retrospective study of 15 patients the term "cicatricial marginal alopecia" was proposed to describe a primary cicatricial hair loss that occurs at the scalp periphery sparing portions of the frontal and occipital hairlines in patients with no history of traction [15]. Half of the patients were Hispanic and had frontal or fronto-temporal hair loss similar to that described in our patients. Histology of patients in this study also revealed decreased numbers of hair follicles, replacement of follicles by fibrous tracts, and intact sebaceous glands. The retention of the sebaceous gland in these patients argues against the diagnosis of a true primary cicatricial alopecia, and points toward a likely diagnosis of late stage traction alopecia. None of the patients gave a history of chronic traction but some subjects did report wearing hair in ponytails. It is likely that these patients, who were on average older than in our series, presented during the later stages of the disease and did not recognize that certain hair care practices earlier in life, could have contributed to their hair loss.

Treatment

Treatment options for TA vary depending on whether or not longstanding disease has resulted in permanent hair loss. Treatment can be divided into three stages: prevention, early TA, and longstanding TA. Prevention is key in childhood and involves educating parents on the importance of loosening the hair-style and avoiding tenting, which occurs when the hair is pulled so tightly that the skin of the scalp is raised by the force of the pull.

... of active inflammation suggesting the disease had progressed to the irreversible phase even though the average age at presentation was relatively young (34 years).

On exam, the characteristic finding of retained but diminutive/smaller caliber hairs along the frontal and/or temporal hairline correlates with the pathologic finding of vellus-sized follicles. The cause for this finding is unclear. It has been proposed that the hairs along the border may be shorter and thus "fall out" of a tight ponytail. An alternative explanation has been that patients with androgenetic alopecia may be more susceptible to TA because of miniaturized hairs. Considering the known plasticity of the hair follicle and the fact that the size of the hair follicle changes over time and is determined by the size of the dermal papilla, it is conceivable that chronic traction may affect the dermal papilla and lead to a diminution of the hair follicle.
In early TA in children it is important to loosen the hairstyle, and avoid chemicals or heat as hair loss is reversible at this stage. Brushing the affected area "to stimulate hair growth" should be avoided. In adults with early TA, the hair-style should also be loosened. In those cases in which ethnic or religious practices do not permit modification of hair-styles, it is important to encourage loosening of the hair-style. Intraleosional triamcinolone, directed at the periphery of hair loss, has been reported to be beneficial in suppressing peri-follicular inflammation in adults with early TA [17]. Oral or topical antibiotics may be used in the early stages of disease for their anti-inflammatory effect [17]. Two percent topical minoxidil has also been reported to promote hair growth in a few patients [18]. In our practice we frequently recommend a trial of minoxidil 5% solution or foam for treatment of TA.

In longstanding disease surgical options may be considered. Hair transplants in the form of micro-grafting, mini-grafting, and follicular unit transplantation have been effective [17,19]. Other options include rotation flaps and scalp reduction [17].

**Conclusion**

Traction alopecia in this cohort of women demonstrated diagnostic challenges which were addressed after careful review of hair care practices, their duration, and correlation with clinical and histopathologic findings. Clinically, the fringe sign can be a useful guide to diagnosis in patients with marginal hair loss, with biopsy used to confirm the diagnosis. Early intervention is vital in order to reverse hair loss in TA as the eventual outcome of hair loss frequently depends on timely diagnosis combined with appropriate counseling of patients.

**References**