ABOUT THE AUTHORS

Renée A. Beach, MD, FRCPC

Dr. Beach is an adjunct Assistant Professor in the University of Toronto's Faculty of Medicine. She completed her medical training at McMaster University (MD) and dermatology residency at the University of Ottawa. She practices medical and cosmetic dermatology at her recently opened practice *DermAtelier on Avenue* in Toronto. At Women's College Hospital, she teaches dermatology residents in the Consultative Hair Loss Clinic.

Kikachukwu Otiono, B.A.
MD Candidate, Michael G. DeGroote School of Medicine, McMaster University (Class of 2022)

Ms. Otiono is a 2nd year medical student at the Michael G. DeGroote School of Medicine at McMaster University. She is a native of Ottawa and the Director of Advocacy & Representation for the Black Medical Students' Association of Canada (BMSAC).

A TALE OF TWO FRONTAL ALOPECIAS: TRACTION ALOPECIA AND FRONTAL FIBROSING ALOPECIA – CLINICAL PEARLS AND PROMISING THERAPIES

Introduction

Traction alopecia (TA) is a form of hair loss caused by repetitive tension and pulling of the hair.[1] TA most commonly occurs in women of Afro-Caribbean descent with afro-textured hair due to the higher prevalence of high-tension hairstyles such as braids and weaves.[2] However, it has also been reported in other patient demographics including ballerinas[3], gymnasts, and patients with certain head coverings.[4] TA typically occurs

in a biphasic nature, starting with an early stage that is non-cicatricial (without scarring) which can progress to cicatricial and permanent alopecia with prolonged tension.[5] Conversely, frontal fibrosing alopecia (FFA) is a variant of lichen planopilaris (LPP) that presents as an inflammatory cicatricial alopecia. FFA is clinically characterized by permanent alopecia, an unpredictable clinical course, and a band-like pattern of involvement in the frontal and temporal hairline.[6] FFA mainly affects post-menopausal women as described by Kossard,[7] although there have been cases reported in premenopausal women and in men.[8]

When hair loss involves the frontotemporal region, it can sometimes be difficult to distinguish between TA and FFA. Marginal or frontal hairline TA can especially mimic the band-like pattern of hair loss seen in FFA. As such, delineation of the clinical presentations and distinguishing features of each condition is important. This paper outlines clinical parameters for each diagnosis to enable accurate diagnosis and therapy.

Clinical Examination

Traction alopecia (TA) is directly related to mechanical trauma and repetitive pulling from high-tension hairstyles, therefore it is crucial to elicit haircare practices during history-taking. Women with afro-textured hair are at highest risk due to the increased prevalence of high-risk hairstyles such as twists, braids, cornrows, weaves which are installed with tension and involve added hair, and dreadlocks which can be heavy as they mature and lengthen.[9,10] Chemical relaxation/ hair straightening products coupled with artificial extensions are also strongly associated with increased risk of TA.[10] This increased risk occurs because the existing hair is permanently weakened, and tension-based styles or added hair further strain the hair strands. It is important to note that although TA is more prevalent in Afro-Caribbean patients, this is more related to common haircare practices rather than afro-textured hair types, as similar haircare practices yield symptomatology in other groups. Examples include tight buns and ponytails, religious practices (e.g. hair twisting in Sikh boys and men), tight scarf styles, extensions,[11] curlers, and chignon.[3]

The clinical features of traction alopecia depend heavily on the level of mechanical trauma and the stage of the disease. The most common presentation of TA is marginal alopecia which affects the frontal and temporal scalp, while the less common nonmarginal alopecia results in hair loss in other areas like the interior scalp.[1] In the earliest stages, patients will admit to sustaining pain or a headache after hairstyle installation. TA is non-scarring and may present as a slight decrease in hair density or small alopecic patches. Among the earliest clinical signs is the presence of traction folliculitis, producing perifollicular erythema, papules, and pustules in areas with the highest tension.[12] The 'fringe sign' is typically present, which is characterized by fine or miniaturized hairs remaining at the margin of the frontal hairline with hair loss posterior to the fringe.[13] There may also be hair casts, which are thin, white-hued cylindrical concretions that surround the hair shaft.[14] Permanent alopecia may occur with persistent mechanical trauma.

Unlike the mechanical pathogenesis of TA, frontal fibrosing alopecia (FFA) is a primary cicatricial alopecia. The pathogenesis of FFA is poorly understood, but LPP in general is believed to occur via an inflammatory process.[15] Overall, LPP seems to be more common in women than in men.[16] The classic presentation of FFA is a band-like hair loss in the frontotemporal hairline in post-menopausal women.[17-19] FFA can also affect premenopausal women, and rarely, men.[6] Multiple studies have also demonstrated a strong association between thyroid disease (especially hypothyroidism) and FFA.[21,22]

Although, the band-like distribution of FFA predominantly occurs in the frontal hairline, scarring can extend to the preauricular and retro-auricular regions of the scalp.[23] It can also affect other areas including the occipital region and the auricular margins.[24] While TA affects only the area of the scalp under tension, FFA commonly causes hair loss in eyebrows as well. In male patients affected by FFA, there may be hair loss in the frontal hairline, eyebrows, beard area and sideburns.[25] Clinicians may also notice a small number of isolated or solitary hairs within the band of alopecia, often referred to as the 'lonely hair sign.'[26] Other clinical features include erythematous and hyperkeratotic follicular openings, hypopigmentation,[27] and trichodynia[28] in patients with white skin. Interestingly, patients with brown skin and afro-textured hair generally display less erythema and may show perifollicular hyperpigmentation instead.

Histopathology

Since TA has a biphasic course, histopathological findings differ based on the severity and stage of alopecia. Early TA is characterized by a normal number of vellus hairs,

trichomalacia, and a decrease in telogen and catagen follicles.[29] Late-stage TA results in scarring with reduced follicular density, retained sebaceous glands, and a decrease in terminal follicle count (follicular 'drop out').[29] Conversely, primary cicatricial alopecias such as FFA feature the replacement of destroyed follicular units with fibrous tissues[30] as well as loss of follicular ostia.[31] FFA is a lymphocytic primary cicatricial alopecia with perifollicular inflammation and lymphocytic cell infiltrate at the infundibulum and isthmus.[32]

Therapeutic Management

Both types of alopecia can be managed according to their clinical severity and progression, as well as patient preference for non-prescription versus prescription-based management **(Table 1)**.

Behavioural Management

For both forms of alopecia, minimizing damage to the frontal scalp is critical. In TA, minimization, if not avoidance, of hair styles with tension or pulling is paramount. Varying scalp parts for typical hairstyles or wearing the hair in a looser style, such as a low bun is helpful.[33] For women and men who wear headgear for religious observance, adjustments like applying a cotton band to the hairline, then the turban or hijab afterwards will help relieve tension. In FFA, there is some association of its provocation following mechanical or thermal trauma to the scalp.[34] Australian dermatologists have proposed an association between FFA and the application of sunscreen to the hairline, including hair regrowth following its stoppage.[35] Therefore, avoidance of these potential triggers at these scalp sites is preferred.

	TA	FFA
Behavioural Measures	Hair style modification Loosen hairstyles or head coverings if painful	Avoid trauma (thermal or chemical burns) to the hairline Apply cosmetic products 2cm away from the hairline
Topical Therapy	minoxidil 5% foam* corticosteroid therapy (medium potency)	minoxidil 5% foam* calcineurin inhibitors* corticosteroid therapy (medium – high potency)
Procedural Therapy	Kenalog injections 5 mg/mL (consider 3 sessions 4 - 8 weeks apart to gauge response)	Kenalog injections 2.5 – 5 mg/mL (performed at 6 – 12 week intervals)
Oral Treatment	Sub-acute duration 3 – 6 months Minoxidil 1.25 – 2.5 mg qhs*	Acute duration ~ 3 mth course with stoppage if clinically stable Doxycycline 100mg twice daily* Sub-acute duration ~ 3 – 6 month course Cyclosporine 150mg twice daily* Chronic duration ~ 6 month course with reassessment of need of prescription continuation Methotrexate 20 – 25mg weekly* Mycophenolate mofetil 1g twice daily* Mycophenolic acid 720mg twice daily* Hydroxychloroquine 5mg/kg/day body weight* Acitretin 10 - 20mg daily Pioglitazone 15mg daily* Finasteride 5 mg daily*
Surgical Treatment	Hair transplant with permanent adoption of low tension hair-styling	Hair transplant if clinically stable and asymptomatic x 12 months or more

Table 1: Management of traction alopecia (TA) and frontal fibrosing alopecia (FFA)
**Off-label therapy*

Topical therapy

In TA and FFA, topical therapy can be used to limit inflammation, particularly in the early stages of the condition, or if there are symptoms of erythema, tenderness, and edema. Use of medium potency steroids like betamethasone valerate 0.1% lotion can help abate clinical symptoms in TA and FFA. Removal of the inciting hairstyle and use of topical minoxidil preparations can stimulate hair regrowth in TA.[36]

Procedural therapy

Reports of hair regrowth following injection of triamcinolone acetonide 5 mg/mL (Kenalog) to the affected scalp in TA make it a feasible in-office therapy at 6-8-week intervals.[37] However, patients risk pain and injection site atrophy. Also, it must be emphasized that this is not curative or a substitution for adaptation of tension-free hairstyling. In FFA, the use of Kenalog therapy at 8–12 week intervals is considered a cornerstone of management. It is postulated to help negate the presence and activity of inflammatory cytokines.[38] There are limited reports of platelet rich plasma (PRP) showing success in FFA for cessation of symptoms and hair regrowth.[39] Patients should be informed of its out-of-pocket cost and potential to require ongoing maintenance sessions to maintain its effect.

Oral Therapy

Although not the cornerstone of therapy, there is evidence that off-label use of oral minoxidil 1.25 – 2.5 mg at night can result in hair regrowth in non-scarred TA patients.[40] Results are noted after 3-6 months of consistent use. Patients should be advised of the risk of extra-scalp hypertrichosis.

There are a range of oral therapies for FFA, many of which are similar to LPP. In the acute phase, short courses of tetracycline antibiotic doxycycline may be helpful in diminishing scalp erythema and inflammatory symptoms. Successful treatment of LPP with immunomodulators such as cyclosporine, methotrexate, and mycophenolate mofetil has been reported and each yield better clinical improvement than hydroxychloroquine.[41] Given its relation to lichen planus, therapy with the retinoid acitretin can be a therapeutic option which also avoids immunosuppression. However, patients should be educated about the potential for telogen effluvium with this treatment. Finasteride treatment can be helpful, perhaps due to concurrent androgenetic alopecia activity. Finally, use of immune-modifiers like pioglitazone have a therapeutic role, particularly for patients who have clinical symptoms associated with their hair loss.[42]

Surgical Treatment

There are successful reports of TA correction with hair transplant in patients with afro-textured hair and straight hair.[43,44] While FFA, if stable for 12 months, can undergo hair transplant, scalp surgery has also been associated with inciting FFA,[45] and thus must be considered as a last resort therapy with a patient who fully understands the potential for a paradoxical result.

Conclusion

These two forms of hair loss, TA and FFA, share a predilection for the frontal scalp and overlapping therapeutic options. Dermatologists' clinical acumen and therapeutic experience are best suited to confirm their diagnoses and optimize therapy. The descriptions of their clinical findings, histological features, and management options provided here are a framework to help provide comprehensive clinical management and improvement.

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