Traction alopecia: A neglected entity in 2017?

Christiana Oyinlola Akingbola, Jui Vyas

Department of Dermatology, School of Medicine, Cardiff University, Cardiff, CF, UK

Abstract

Traction alopecia was first described in 1904 but is still a cause of scarring hair loss in young women worldwide. It is unique in being initially a reversible then an irreversible (scarring) form of alopecia. Linked to tightly-pulled hairstyles, it is seen across all races. The pattern of hair loss depends on the style creating it but most commonly affects the frontotemporal hairline. There are some new examination findings associated with traction alopecia, which are traction folliculitis, the fringe sign and hair casts (pseudonits) on dermatoscopy. These may prove key in prompting early specialist referral. The mainstay of current treatment is cessation of the contributing hairstyles. Camouflage, anti-inflammatory or growth-stimulating topical preparations are second line treatments. In later stages of severe traction alopecia hair transplantation may be the only effective treatment. The evidence basis for medical intervention with topical agents is anecdotal at best. Furthermore, additional research is required to clarify the pathogenesis of this biphasic alopecia. Until then, prompt diagnosis and identification of causative hairstyles are focus of current dermatological practice.

Key words: Alopecia, hair loss, scarring alopecia, traction alopecia, traction hair loss

Correspondence:

Dr. Christiana Oyinlola Akingbola, Department of Dermatology, School of Medicine, Cardiff University, Cardiff, CF, UK. E-mail: lolascrib@gmail.com

Introduction

Traction alopecia is caused by persistent, pulling forces on the hair follicles from traction-inducing hairstyles. It has been reported across all races.¹⁻³ Traction alopecia uniquely exists in both the scarring (cicatricial) and non-scarring alopecia categories depending on whether it is early and reversible or late and permanent. The greatest risk from a delay in diagnosis is the progression to scarring traction alopecia.

This review will underline the current understanding of traction alopecia's pathogenesis, its management and their evidence bases.

Epidemiology

At present, traction alopecia has been described across most races. Examples include young Sikh men with tight knots beneath their turbans,¹ a Caucasian ballerina wearing tight buns for 13 years² and Hispanic women with tight ponytails⁴. The prevalence of traction alopecia in different populations is still uncertain.

Two South African studies by Khumalo *et al.* found that $31.7\%^5$ of women and $9.4\%^6$ of children had traction alopecia. They also showed a higher prevalence with increasing age in 8.6% of younger

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girls (ages 6–7) and 21.7% of older girls (ages 17–21).⁶ In contrast, a Nigerian study found traction alopecia in only 7.7% (3/39) of women.⁷ Wright *et al.* (an American study) quoted traction alopecia in 18.4% (37/201) in a questionnaire-based study in young girls (ages 1–15).⁸ Unfortunately, the Nigerian and American studies had small sample sizes and very little detail of their clinical and histological criteria. Furthermore, there were potential confounders of pregnancy, malnourishment and recall bias about which specific hairstyles were worn regularly. Interestingly, the American study relied solely on carer- or parent-based diagnoses of traction alopecia. In contrast, with their larger sample sizes and dermatologist or experienced clinician diagnoses, the South African studies had more robust findings.

Nonetheless, these studies all agree that traction alopecia is frequently seen in women and children of African descent with a greater prevalence noted with increasing age. They also underline the scarcity of prevalence data from other populations.

Diagnosis

The three key steps for diagnosing traction alopecia are history-taking, scalp examination and specialist tests.

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Pertinent questions in the history

These include the duration of the hair loss, which styling practices were previously employed and traction symptoms while creating such hairstyles. In 2008, Khumalo *et al.* found that traction alopecia was almost twice as likely to be present in South African women or children who had previously experienced tight, painful braids (odds ratio [OR] =1.94 [P = 0.004]) or noted "pimples" at the base of their braids (OR = 1.81 [P = 0.046]) at the time of styling, both of which were statistically significant.³ This suggests a link between signs of traction during hairstyling and diagnosable traction alopecia.

Clinical examination

This begins at the scalp by looking at the area of hair loss and the hair strands. The hairstyle of the patient is also important. Below are signs that help strengthen a diagnosis of traction alopecia.

Traction folliculitis

Fox et al. first described this perifollicular erythema (redness) and/or pustules following long-standing traction on the hair strands.9 They noted that these changes were found in the areas that were under maximal tension from the hairstyle.9 Later, Puttgen reported signs of a dry, itchy and flaky scalp and proposed that the pustules were usually sterile.¹⁰ Urbina et al. also presented a case report of six Chilean ladies (aged 12-26) with traction folliculitis after wearing tight braids, glued-on hair extensions and ponytails.¹¹ These resolved with traction-avoidance advice and oral antibiotics (though only two cases were confirmed to have Staphylococcus aureus). Interestingly, such findings were described by Hjorth in 1957 as "groups of inflamed follicles," "perifollicular erythema" and "firmly adherent crusts or scales suggestive of seborrheic eczema" in his marginal traction alopecia case report.¹² Lastly, Khumalo et al. also found that 24.3% (150/604) of South African women with tightly braided hairstyles reported symptoms and signs suggestive of traction folliculitis.5 Discovering the true prevalence of traction folliculitis would require larger, multi-centered surveys with various racial backgrounds included. An example of traction folliculitis is given here [Figure 1].

Pattern of hair loss

Traction alopecia typically affects the frontotemporal margins of the scalp. However, the specific causative hairstyle will determine any other pattern of hair loss seen.¹²⁻¹⁸ Within areas of hair loss, there



Figure 1: Traction folliculitis. Perifollicular erythema (blue arrows)

will be thin, vellus-like hairs centrally with surrounding mid-length thicker hairs (broken terminal hairs).¹³ Table 1 summarizes the various patterns of hair loss (Adapted from Whiting¹⁴ Hantash and Schwartz¹⁵, Costa¹⁸ and Ahdout and Mirmirani¹⁷). Figure 2 shows posterior margin traction alopecia.

The "Fringe Sign"

This sign, first suggested in 2011 by Samrao *et al.* describes short terminal hairs bordering areas of marginal hair loss.⁴ Interestingly,

Table 1: Patterns of hair loss			
Pattern of hair loss	Causative style	Differential diagnosis	
Ophiasiform: Bitemporal, above ears, frontal margin, nuchal area	Ponytails and long braids	Ophiasis-pattern alopecia areata	
Frontoparietal hair loss	Twisting long hair (Sikh boys)		
Frontotemporal hair loss	Hair rollers	Frontal fibrosing alopecia	
Frontal hair loss only	Tight scarves (religious reasons)	Frontal fibrosing alopecia	
Central "V" parting hair loss	Tight plaiting of hair		
Crown hair loss	Hair weaving (weft attachment)		
Occipital hair loss	Chignons (where chignon rests)		
"Horseshoe" – semicircle from temple, through crown to temple	Repeated glued-on weft of hair		

Adapted from Whiting, Hantash and Schwartz, Costa and Ahdout and Mirmirani



Figure 2: Posterior hairline traction alopecia



Figure 3: The fringe sign at the right frontotemporal hair margin

it is present before clinical hair loss and persists throughout to late traction alopecia.⁴ Of note, Costa¹⁸ and Hjorth¹² similarly described this "strip of peripheral hair" in 1946 and "a thin straggling strip of hair at the distal margin" in 1957. The historical constancy of this sign lends credibility to its use as a clinical marker of traction alopecia. In addition, it rules out the main differentials of ophiasis pattern alopecia areata and frontal fibrosing alopecia, respectively [Figure 3].⁴

Supplementary tests

Medical photography

This aims to accurately record the extent of traction alopecia and the response to treatment. Khumalo *et al.* developed the Marginal Traction Alopecia Severity Score in 2007, using photographs of the anterior and posterior hair margins.¹⁹ While it showed good inter-observer agreement in the photograph-matched severity scores (interclass correlation coefficient = 0.99), it was unreliable for diagnosing posterior margin traction alopecia.¹⁹ It still needs to be validated for use in studies and trials.

Dermatoscopy

With its magnified images, dermatoscopy (dermoscopy) allows finer details to be appreciated than naked eye examination.²⁰ As a non-invasive technique, it should be integral to clinical examination



Figure 4: Dermatosopic Image x 20 magnification. Hair casts (yellow arrows), vellus like hair (blue arrows), pinpoint white dots (red arrows). Image courtesy of Dr Antonella Tosti

Table 2: Changes seen in early and late traction alopecia			
Features on biopsy	Early TA	Late TA	
Terminal hair density	Reduced	Markedly reduced	
Perifollicular appearance	Mild or no inflammation	Fibrous tracts (scarring)	
Vellus-like hairs	Preserved	Increased	
Hair cycle changes	Increased nonanagen hairs	"Follicular dropout"	
Sebaceous glands	Preserved	Preserved	
Adapted from the lite	rature. TA: Traction alopecia		

of the scalp. Yin and Tosti suggest using it as a guide for choosing the biopsy site.²¹ Dermatoscopy in traction alopecia shows broken hairs, miniaturized hairs, pin-point white dots (loss of follicular openings or ostia), reduced hair density and hair casts.^{21,22}

Hair casts

Hair casts (pseudonits) are 2–7 mm long, freely moveable, gray-white cylinders wrapped around the proximal hair shaft of hairs surrounding areas under tension.^{21,23} They were first associated with traction-inducing hairstyles in 1990 by Zhu *et al.*²⁴ Zhang confirmed this in 1995 with a prospective survey of 3548 school-aged women and girls where 81% (1073/1332) of women and girls with tightly braided hair exhibited this sign in the areas under maximal tension.²⁵ Recent case reports by Tosti *et al.*²⁶ and Ozuguz *et al.*²⁷ have reinforced this finding [Figure 4].

Histopathology of traction alopecia

Traction alopecia staging is based on its histological appearance as seen in Table 2 (adapted from the Literature^{2,26-33})and the histology slides in Figures 5 and 6. Unlike other causes of scarring alopecia that show perifollicular fibrosis and inflammatory infiltrates with loss of sebaceous glands, traction alopecia has preserved sebaceous glands, increased vellus-like hairs and paucity of inflammation.^{2,28}

Follicular miniaturization

In 2008, Khumalo *et al.* hypothesized that the presence of short velluslike hairs in early traction alopecia may lend credence to a follicular miniaturization theory.³ This was supported in 2012 by Miteva and Tosti's findings of miniaturized follicles (diameter <0.03 mm) in scalp biopsies [Figure 5] of African-American women with traction alopecia²⁹ and its persistence to late-stage traction alopecia. Not surprisingly, they highlighted the possible misdiagnosis of such findings as androgenetic alopecia in the absence of scarring changes in early traction alopecia. Goldberg suggested a means of circumventing this obstacle by noting the overall reduced follicular density of the specimen.¹³ He found only 7–8 follicles (on average) per 4 mm punch graft specimen in traction alopecia compared to normal ranges of 21–38 follicles in his 15 biopsy specimens.¹³ Nonetheless, this step in the pathogenesis of traction alopecia requires more research to determine if the miniaturization process is a viable target for future therapeutic options.

Follicular drop-out late-stage (scarring) traction alopecia

In this stage of traction alopecia, there are reduced follicular numbers with minimal or no surrounding inflammation.²⁹ The presence of fibrous tracts indicates a loss of the hair follicles (follicular dropout). In further contrast to other forms of scarring alopecia, the sebaceous glands are preserved [Figure 6].^{28,31-33} The point at which the above changes occur is yet to be clarified.

Current Management

The treatment of traction alopecia begins with conservative measures and progresses through medical and surgical interventions.

Conservative management

The mainstay of management is the cessation of the traction-inducing hair practices. This is through patient education with clear guidance



Figure 5: Horizontal section of early traction alopecia with vellus-like follicles (yellow arrows) and retained sebaceous glands (black arrows) (H and E, x10). Image from Miteva and Tosti 2012^{29} . Copyright 2012 by John Wiley and Sons

on looser hairstyles and the avoidance of chemical damage. In 2014, Mirmirani and Khumalo noted that caregivers (parents) and hairdressers should be primary targets for any educational effort as children and adolescents seemed most at risk.³⁴

Although wigs and scalp camouflage (sprays and lotions) help hide areas of hair loss, hair pieces or extensions are discouraged. This advice followed case reports of worsening traction alopecia in a 17-year-old ballerina² and a "horseshoe" pattern of hair loss in two women with glued-in hair pieces.¹⁷

Medical management

Anti-inflammatory preparations

Oral and topical antibiotics are recommended for treating traction folliculitis for their anti-inflammatory effects.^{9,11,34} Although some articles have mentioned the anecdotal use of corticosteroids (either topically or intralesionally) in traction alopecia, there were no clear cases demonstrating the benefit of this treatment in the literature.^{34,35}

Topical minoxidil 2% preparations

Although mainly used in androgenetic alopecia where it stimulates follicles to differentiate, minoxidil has been discretely used in traction alopecia. In a case report by Khumalo and Ngwanya, traction alopecia refractory to cessation of traction-inducing practices for up to 2 years regrew after 3 months of treatment with 2% minoxidil in two women with long-standing disease.³⁶ It is also routinely used in the peri- and post-operative period of hair transplantation to reduce graft loss and encourage regrowth.³⁵

Surgical management

In refractory cases of late traction alopecia, surgical options include punch grafting, micro- or mini-grafting and follicular unit transplants.³⁷ The choice of technique is tailored to the patient and the pattern of hair loss. Punch graft hair transplants, first described in 1959, involve transferring 2–4 mm round grafts to the area of alopecia.³⁵ Follicular unit transplantation has superseded this technique. A microscopically dissected follicular unit - one to four follicles growing in a bundle – is implanted at the recipient site.³⁷ Advantages of the latter include the seamless appearance of the transplanted hairline, the ability to move more follicular units and the smaller wound size.³⁸

Callender *et al.* found that follicular unit transplants seemed more suited to straighter Caucasoid hair strands while mini- and punch-grafting were better in those of African descent.³⁵ They postulated that the latter's curved follicles were more likely to be transected (sliced) during follicular unit extraction. Bernstein *et al.* reported that this occurred in 1.7%–15% of such patients.³⁸



Figure 6: Horizontal section of late-stage traction alopecia with preserved sebaceous glands and fibrous tracts (H and E, $\times 20$) (Image courtesy of Dr. Kenneth May)



Figure 7: Diagram showing predisposition of curly African hair (left) to traction damage along its length versus straight Caucasian hair (right). (Points of weakness in red). Image from Miteva and Tosti, 2012²⁹ Copyright 2012 by John Wiley and Sons

However, the curly strands are purported to give a fuller-looking result using the traditional methods.³⁵ The lack of statistical figures or analyses to support this assertion does limit its credence. Careful patient selection is needed to minimize the risk of hypertrophic and keloid scarring when considering hair transplantation.

Discussion

After reviewing the literature, it is clear that traction alopecia is not a new condition. It progresses from a pre-alopecic, inflammatory stage that includes traction folliculitis, through a reversible alopecia (early traction alopecia) to a scarring stage (late traction alopecia) – as seen in Figure 8. Such a timeline may allow earlier intervention with medical therapies. Should these be successful, they would be clues to the pathogenesis from traction to inflammation and eventual scarring [Figure 8].

The high prevalence rates of traction alopecia noted in those of African descent infers either a genetic susceptibility or persistent environmental factors not addressed by current clinical practice.

Genetic susceptibility to traction alopecia

This genetic susceptibility may be due to the asymmetrical shape of the African hair follicle. It has a retrocurvature at the hair bulb and an S-shaped hair shaft. Thibaut *et al.* argued that this shape creates geometric points of weakness along the hair shaft.³⁹ Coupled with fewer anchoring fibers within the dermis, it may predispose them to greater traction forces as shown in Figure 7.⁴⁰

Environmental contributors to traction alopecia

There are two main environmental factors for people of African descent: the chemical processing or "relaxing" of hair strands to achieve a straight appearance and braiding or twisting practices.³⁴

"Relaxers" have been in use since the early 1900s to straighten African hair by breaking disulfide and hydrogen bonds within the strands. Research has shown that relaxed hair is more fragile and probably more prone to tension forces.^{5,6} With 59% of South African girls⁶ and 80% of African-American women⁴¹ chemically straightening their hair, this may be a key factor in the development of traction alopecia.

In 2014, Mirmirani and Khumalo have proposed a sign to alert women to high tension levels in their braided or twisted hairstyles:



Figure 8: A revised probable pathogenesis of traction alopecia with genetic and environmental factors

"tenting."³⁴ This denotes a raised scalp or bumps from the excessive pull on the hair in a braided or twisted style.³⁴ Hairstyles eliciting this sign should be undone to prevent hair loss.

Finally, the psychosocial impact of traction alopecia has not been explored in the literature. Too often, the physician's focus in making treatment decisions is on the clinical severity of hair loss as evidenced by tools like the marginal traction alopecia severity score. However, it has been shown that physician-perceived hair loss severity does not correlate with the patient's hair loss severity or its effect on their quality of life.⁴² Accordingly, patients who felt their disease was more severe or they had no control over it had poorer quality of life outcomes.⁴² Furthermore, Cash found that patients seeking professional consult have usually tried several coping mechanisms (like camouflage) before presenting.⁴³ Various quality of life indices have been trialed including the Dermatology Life Quality Index, Skindex and Scalpdex.⁴² These tools can be filled by patients on their first and subsequent consultations.

By exploring the patient's concerns regarding traction alopecia and its regrowth potential, physicians could improve the compliance with medical advice and therapies.

Conclusion

Although traction alopecia has long been agreed to be the end point of long-standing tension hairstyles, steps in its pathogenesis are still perplexing. The presence of vellus-like hairs throughout its development points to follicular miniaturization as a yet under-explored aspect of its pathogenesis. Furthermore, the progression from a reversible hair loss to scarring alopecia is still poorly understood. Nonetheless, traction folliculitis may represent the earliest point for medical intervention. Moreover, newer therapies targeting miniaturized follicles as in androgenetic alopecia may in turn revolutionize traction alopecia management.

Finally, a more robust evidence base for the current medical therapies will hopefully produce a comprehensive treatment algorithm for the optimal management of traction alopecia.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patients have given their consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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