TRACTION ALOPECIA: A REVIEW

INTRODUCTION

Traction alopecia (TA) is a term used to describe hair loss caused by prolonged or repetitive tension on the hair. Traction alopecia was first described in 1907 in subjects from Greenland who developed hair loss along the hairline because of the prolonged wearing of tight ponytails. Subsequently, most of the literature has focused on the prevalence of TA in people of African descent. However, TA affects people of all different ethnic backgrounds and is the result of an individual’s hair care practices. It 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. Because pathologic features are biphasic and show varying features in early and late stage disease, appropriate clinical-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 diagnosis is broad and can include alopecia areata, androgenetic alopecia, telogen effluvium, trichotillomania, and primary lymphocytic cicatricial alopecias (lichen planopilaris, central centrifugal cicatricial alopecia, pseudopelade of Brocq, and frontal fibrous alopecia). Traction alopecia of the marginal hairline may be misdiagnosed as ophiasis pattern alopecia areata or frontal fibrosing alopecia because these disorders can have a similar band-like or patchy pattern of hair loss. 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 Sikh males as a result of twisting their uncut hair tightly on the scalp. Submandibular TA has also been reported in a Sikh male who tied his beard in a tight knot below the chin. 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. 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. Traction alopecia 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. 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. The frequency of hair relaxing, however, does not appear to affect susceptibility of developing TA. Chemically processed hair may be less resistant to TA than natural hair. Patients who develop any symptoms with hairdressing (including pain, pimples, stinging, or crusts) have also been shown to be at increased risk of developing TA. 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 of the marginal hairline. 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.

**Histopathology**

**Figure 5:** Chronic traction alopecia. Scalp biopsy from patient 1 shows (5a) retention of sebaceous glands (H&E, x40) and (5b) fibrotic fibrous tracts (H&E, x100). These findings are consistent with chronic traction alopecia.

**Figure 6:** (6a) Biopsy from a patient with traction alopecia reveals a slightly reduced follicular density, follicular miniaturization, and retained sebaceous glands (H&E, x40). (6b) Follicular miniaturization and retained sebaceous glands are observed (H&E, x100). (6c) Many fibrotic fibrous tracts (lower half) are present (H&E, x200).
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. With longstanding TA, sebaceous glands are present but may be decreased and vellus-sized hairs may be seen (Figures 6a and 6b). There is a decrease in the number of terminal follicles, which are replaced with fibrotic fibrous tracts (Figure 6c). 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, histopathology revealed findings consistent with the diagnosis of longstanding TA (Figure 5). 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 of mostly late stage findings on biopsy specimens. An additional factor may have been a bias in the clinic to more frequently biopsy patients with late stage disease or a remote history of traction.

Causes of Traction Alopecia

- Telogen conversion
- Long term hair pulling and breakage due to very tight hair braiding, hair weaves and cornrows
- Over use of hair softeners such as permanent wave solutions
- Repetitive application of hot combs
- Chemical treatment of hair with dyes, bleaches, or straighteners

Following are the top ten causes of traction alopecia.

1. Drawstring ponytails

These hair pieces have a comb and drawstring to secure them on top of the head. Drawstring ponytails are attached to the hair after the hair has been pulled back into a tight bun. Hair gel is sometimes applied to achieve a sleek and smooth finish to the natural hair that is held in a bun. The drawstring ponytail is then attached by pinning it in place with the comb and using the drawstring to secure it. Although they are a convenient way to style your hair, frequent and constant use can cause traction alopecia, particularly in the area where the ponytail is attached.

2. Tight buns

The hair is twisted, rolled tightly and then fastened with pins or ties. The continuous effect of twisting and rolling the hair can weaken the strands and damage the follicles, resulting in traction alopecia.

3. Weaves

This is a style very popular (but not restricted to) among black women and involves human or synthetic hair wefts being attached to natural hair, often sewing it on to cornrowed tracks. Wefts are sometimes also attached by using an anti fungus adhesive called bonding glue. Weaving is often used to stop the appearance of thinning hair, but unfortunately it can also cause thinning and hair loss itself, as the cornrows the extensions are attached to are very tight to make the weave last longer. Bonding glue can also
cause hair loss when a proper remover isn’t used to break the glue’s bond completely before removal.

4. Braids

Hair is braided into thin, tight braids, sometimes with the addition of decorative items or with hair extensions braided into the hair (remember Brandy’s signature braids?). Apart from the pulling action caused by the tight braid, the hair line suffers because the hairs the extensions are attached to are usually weak and not able to cope with the weight of the additional hair.

5. Cornrows

These are a type of braids where the hair is braided close to the scalp. This style is favoured for being a low maintenance, aesthetic hairstyle, but can lead to traction alopecia if the cornrows are too tight as they place undue pressure on the hair, especially around the hairline.

6. Clip on hair extensions

These hair extensions are made by cutting machine made hair wefts into different lengths and attaching clips to each piece. They can be clipped onto the natural hair in various places, including the back, the sides of the face, etc.. They are commonly used to add colour to the hair or to give the appearance of fuller hair. Improper application by clipping the extensions too tightly or allowing the clips to dig into your scalp constantly when applied can lead to traction alopecia.

7. Weight of hair extensions

Hair extensions can be glued or woven onto the hair but either way will increase the tension of the natural hair and the weight of the hair. It’s important to avoid using too much hair to avoid this tension ultimately leading to thinning and hair loss.

8. Tight sleeping caps and scarves

Often worn to maintain hairstyles while sleeping, tight sleeping caps and scarves can restrict blood flow to the area its tied around, depriving the hair of essential nutrients and slowly suffocating the follicle.

9. Sleeping in rollers

Rollers are used for curling the hair and many women wear them in bed, as the curling process can take a significant amount of time to work. Tight rollers can cause the hair to fall out in clumps.
10. Turbans

This is a cause that mostly affects Sikh men. When the turban is worn too tightly, it can cut off the blood supply to the follicles, resulting in hair thinning and hair loss.

Pathophysiology

Hair follicle growth occurs in cycles. Each cycle consists of a long growing phase (anagen), a short transitional phase (catagen) and a short resting phase (telogen). At the end of the resting phase, the hair falls out (exogen) and a new hair starts growing in the follicle beginning the cycle again. Normally about 100 hairs reach the end of their resting phase each day and fall out. When more than 100 hairs fall out per day, clinical hair loss (telogen effluvium) may occur. A disruption of the growing phase causes abnormal loss of anagen hairs (anagen effluvium).

Symptoms of Traction Alopecia

- Itching and dandruff.
- Cicatrization (scarring) of the new hair follicle and permanent hair loss.
- Patchy areas of hair loss.
- Perifollicular erythema, scales, and pustules on scalp.

Diagnosis

Evaluation for causative disorders should be done based on clinical symptoms. Because they are not usually associated with an increased loss rate, male-pattern and female-pattern hair loss don’t generally require testing. If hair loss occurs in a young man with no family history, the physician should question the patient on drug and illicit drug use.

The pull test: this test helps to evaluate diffuse scalp hair loss. Gentle traction is exerted on a group of hair (about 40–60) on three different areas of the scalp. The number of extracted hairs is counted and examined under a microscope. Normally, <3 hairs per area should come out with each pull. If >10 hairs are obtained, the pull test is considered positive.

The pluck test: In this test, the individual pulls hair out “by the roots.” The root of the plucked hair is examined under a microscope to determine the phase of growth and used to diagnose a defect of telogen, anagen, or systemic disease. Telogen hairs are hairs that have tiny bulbs without sheaths at their roots. Telogen effluvium shows an increased percentage of hairs upon examination. Anagen hairs are hairs that have sheaths attached to their roots. Anagen effluvium shows a decrease in telogen-phase hairs and an increased number of broken hairs.

Scalp biopsy: This test is done when alopecia is present, but the diagnosis is unsure. The biopsy allows for differing between scarring and nonscarring forms. Hair samples are taken from areas of inflammation, usually around the border of the bald patch.

Daily Hair Counts: This is normally done when the pull test is negative. It is done by counting the number of hairs lost. The hair that should be counted are the hairs from the first morning combing or during washing. The hair is collected in a clear plastic bag for 14 days. The strands are recorded. If the hair count is >100/day, it is considered abnormal except after shampooing, where hair counts will be up to 250 and be normal.

Trichoscopy: Trichoscopy is a non-invasive method of examining hair and scalp. The test may be performed with the use of a handheld dermoscope or a video dermoscope. It allows differential diagnosis of hair loss in most cases. 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. The biopsy was taken from the margin of the alopecic patch. 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 (0.03 mm), fibrotic fibrous tracts, and the presence of inflammation.

**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 long-standing TA. Prevention is key in childhood and involves educating parents on the importance of loosening the hairstyle 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.

The toolkit should include both topical treatments, supplements and scalp circulation boosting techniques like massage. Incorporating hair loss shampoos and tonics into your hair care regime during and post treatment can be beneficial in increasing circulation and strengthening your newly grown hair. The hair loss treatments listed below have shown positive results in the treatment of traction alopecia

- Topical or oral antibiotics to prevent inflammation and infection
- Surgical hair transplantation procedures such as punch grafting, flap rotation
- Diet with sufficient levels of iron and protein for normal hair growth

**External treatments for traction alopecia**

Treating hair loss directly at the source, external treatments are available in topical form and are generally considered to yield the most immediate and noticeable effects.

**Minoxidil**

This is a non-prescription medication approved for androgenetic alopecia and alopecia areata. Minoxidil comes in a liquid or foam that is rubbed into the scalp twice a day. This is the most effective method to treat male-pattern and female-pattern hair loss. However, only 30–40% of patients experience hair growth. Minoxidil is not effective for other causes of hair loss except alopecia areata. Hair regrowth can take 8 to 12 months. Treatment is continued indefinitely because if the treatment is stopped, hair loss resumes again. Most frequent side effects are mild scalp irritation, allergic contact dermatitis, and increased facial hair.

**Minoxidil: proposed mechanisms of action**

- Vasodilatory properties
- Angiogenic properties
- Enhanced cell proliferation and DNA synthesis
- Potassium channel opener
- Antiandrogen effects
- Suppression of collagen synthesis
- Immunosuppressive effects

One case report described how two African American patients, 45 and 54 years of age, respectively, found that their frontal hairlines improved after 3 to 9 months of topical 2% minoxidil. Although other treatments such as intralesional steroid injections are considered first-line treatment, we frequently include topical minoxidil in the treatment of this group, especially when hair loss is still in its early stages.

**Pharmacokinetics**

Studies on percutaneous absorption found that twice daily application of 1% to 5% topical minoxidil to the bald scalp corresponded to an average systemic dose of 2.4 to 5.4 mg/day. With topical application, the serum concentration of minoxidil rarely exceeds 5 g/L, and is frequently even below detectable levels. In one study, only 7 of 12 AA patients had detectable levels, ranging from 0.4 to 7.5 ng/mL. Another showed that serum concentration was fairly constant at 2, 4, 6, 15, and 24 hours after a single application (averaging 15 ng/mL with 5% minoxidil). The elimination half-life of minoxidil is 3 to 4 hours, suggesting that the medication is cleared 12 to 20 hours after application. The oral form of minoxidil is metabolized 90% in the liver, mostly by conjugation with glucuronic acid. It is minimally protein-bound and readily excreted by the kidney. After a topical application of radio-labelled 1% to 5% minoxidil daily for 9 days, mean urinary recovery was less than 5% of the administered dose, with no radioactivity found.
in fecal samples. Unfortunately, discontinuation of the drug does indeed result in the loss of recruited hairs. Four out of 10 men with male pattern baldness on 2% or 3% minoxidil for 4 months had nonvellus hair counts that even fell below baseline levels after stopping the drug.

**Side effects**

Some women may have hair follicles that are more sensitive to minoxidil and thus should start with the lower strength (2%). Other women with hyperandrogenism may already have hirsutism that is enhanced by minoxidil. Likewise, some women by ethnicity may have hypertrichosis even before starting minoxidil therapy. It is not fair to tell these women that they cannot use minoxidil, but it is wise to use a lower dose and perhaps take a baseline photograph of their forearms or other areas.

Regaine brands but also available in equally effective generic brands (such as Kirkland), minoxidil is a topical treatment that grows hair only at the site of application. Recommended use is for 5% concentration applied twice a day to the target area. For those with particularly sensitive scalps, a new foam-based version of Regaine is reported by users to be less irritating. Results should be seen within three months, though users are cautioned to continue to use a healthy hair regimen and avoid overly stressing or pulling the hair. Hands should always be washed before and after applying minoxidil. For women worried about potential facial peach fuzz, this occasional side effect will disappear once use is discontinued.

**Finasteride**

This decreases dihydrotestosterone levels, producing an increased amount associated with hair covering more of the scalp. "A small go through suggested that Finasteride worked well better by 2% than Minoxidil, and that there may also have been an improvement with combination therapy. Clinical observation also suggests that it can be easier to use. The relationship of baldness with testosterone levels was observed by Hippocrates, who noticed that young male eunuchs did not develop hair loss. Male pattern baldness also does not occur in men with a genetic deficiency of the second isoenzyme. Both types I and II of 5-alpha reductase convert testosterone to dihydrotestosterone (DHT). Type I predominates in the skin, including the scalp, while type II is present in hair follicles and the prostate. The finasteride molecule works by inhibiting type II 5-alpha reductase. This lowers serum and scalp levels of DHT while increasing scalp levels of testosterone. The effect of finasteride on scalp and serum hormone levels. These effects on scalp and serum DHT and testosterone levels were demonstrated in 17 patients who underwent scalp biopsy before and after a 28- day treatment with either placebo or finasteride 5 mg daily. At baseline, DHT levels were higher in balder areas of the scalp compared to areas of the scalp that had hair, but there was no difference in testosterone levels.

**Side effects**

Besides the risk of feminization of a male fetus, there have been scattered reports of sexual side effects. Approximately 1 in 50 males (2%) reported one or more sexual side effects during finasteride (decreased libido, erectile dysfunction, or ejaculation disorder), compared with 1% in the placebo group. Gynecomastia and other breast disorders, such as mastalgia, were reported in 0.4% of patients, and did not occur until later in the treatment period. More unusual side effects were exfoliative dermatitis, perioral numbness, and swollen glands, all of which resolved with drug cessation and returned with rechallenge.

**Dutasteride**

Dutasteride shares important characteristics with finasteride. While finasteride inhibits type II 5-a reductase, dutasteride inhibits both types I and II 5-a reductase isoenzymes. There is no isolated genetic deficiency of type I 5-a reductase to assess its role in male pattern hair loss. However, there is evidence that dutasteride is three times as potent as finasteride at inhibiting type II 5-a reductase and more than 100 times...
as potent at inhibiting the type I enzyme. This suggests enhanced efficacy over the existing finasteride. Because of these increased effects on the 5-a reductase enzymes, scalp and serum levels of DHT are more affected. Dutasteride can decrease serum DHT by more than 90%, while finasteride decreases serum DHT by 70%. One 4-year study of men on dutasteride 0.5 mg continuously for BPH showed a near complete suppression of serum DHT, decreasing by a mean of 93% from baseline. In comparison with dutasteride, finasteride reportedly reduces scalp DHT by only 34% to 41%. As with finasteride, inhibition of the 5-a reductase enzyme can increase levels of testosterone locally in the scalp. However, the increased efficacy means that it can also increase testosterone levels in the serum. The 4-year study above noted that serum testosterone rose by 25% from 3951.9 pg/mL to 4767.0 pg/mL. Minimal dose-dependent effects on serum testosterone have been described for finasteride. An overview of these effects is provided earlier in Table III.

Efficacy
Dutasteride was approved by the FDA in October 2002 for the treatment of symptomatic BPH at dosage of 0.5 mg daily. It is manufactured by GlaxoSmithKline (New York, NY), and comes in soft gelatin capsules under the trade name Avodart. So far, few studies have been done to assess its efficacy in the setting of hair growth. Phase II trials in 416 men showed that dutasteride increased scalp hair growth in a dosedependent fashion (0.05/0.1/0.5/2.5), and that the dutasteride 2.5 mg group was superior to the finasteride 5 mg group at both 12 and 24 weeks in increasing target hair growths. One very interesting case report describes a 46- year-old woman who had only limited improvement from finasteride who was placed on dutasteride and an OCP. After 6 months of 0.5 mg daily, she had significant thickening of hair shafts, as noted on dermoscopy. By 9 months, the clinical diagnosis of AGA could no longer be made. Another study in 17 sets of male twins with AGA showed that after 1 year of dutasteride 0.5 mg daily, the treatment group had significantly more hair regrowth than did the placebo group. Nonetheless, phase III FDA trials appear to be on hold for using dutasteride to treat male pattern hair loss.

Side effects
As with finasteride, women who are pregnant or thinking of becoming pregnant should not consume or handle this medication because of the potential feminizing effects on a male fetus. One study noted a decreased libido in two of 70 patients for both the 0.05- and 0.1- mg dutasteride groups, and nine of 71 patients treated with dutasteride 2.5 mg for 24 weeks. This compared with three out of 70 patients treated with finasteride subjects and two of 64 placebo patients. Impotence occurred in only two patients taking dutasteride 0.05 mg, one patient taking finasteride 5 mg, and three members of the placebo group. Gynecomastia developed in only one patient in the placebo group.

Other antiandrogen therapies
Some women with hair loss have a spectrum of other symptoms, including hirsutism, irregular periods, and acne. It is important to look for these signs. Patients who are of normal weight and pluck, shave, or bleach any unwanted hair will be harder to recognize. Such patients may also suffer from polycystic ovary syndrome. They may also suffer from androgen-producing tumors of the ovaries or adrenal glands or congenital adrenal hyperplasia. It is important to first ask patients whether they have had laboratory tests for changes in their hormone levels. If not, one may consider referral to an endocrinologist to understand the subtle changes. Many other laboratory studies may be performed, such as a thyroid panel and iron studies, but they are beyond the scope of this discussion. Sinclair investigated the treatment of female pattern hair loss with oral antiandrogens. In an openlabel study, spironolactone 200 mg/day was administered to 40 women and cyproterone acetate to another 40 women. The results showed no significant difference between the two groups, so the results were combined. Overall, 44% of women (35/80) had hair regrowth, 44% (35/80) had no clear change, and 10 women (12%) had continuing hair loss. Logistic regression found no predictors of response among such factors as patient age,
menopause status, serum ferritin, serum hormone levels, severity of hair loss, or histologic parameters. In another study, cyproterone acetate was compared to minoxidil to assess its efficacy in the treatment of female pattern hair loss. A trial of 66 women were randomized to take cyproterone acetate 52 mg daily plus an ethinyl estradiol contraceptive or to apply minoxidil 2% twice daily plus a combined OCP. The results showed a mean increase in hair growth for minoxidil group and mean decrease for the cyproterone group. However, they believed that the minoxidil worked better in patients without signs of hyperandrogenism, and that cyproterone worked better in patients who did have those signs. These results are promising, but must be further investigated in larger, controlled studies. It is unlikely that either spironolactone or cyproterone acetate will ever show more efficacy than minoxidil, which is the current standard of treatment for hair loss in women. In the event that spironolactone is used, recall that it is pregnancy category D. Patients should ideally be on birth control, and they should be reminded that the drug may elevate potassium levels. Flutamide is another medication that has been used to treat hirsutism, but so far there is little evidence to show its effect in regrowing hair.

**Ketoconazole**

Ketoconazole is an imidazole antifungal which has been found to be effective in the treatment of seborrheic dermatitis. One open-label study of minoxidil 2% with ketoconazole 2% shampoo for AGA in men showed comparable growth in both groups, with both achieving better growth than unmedicated shampoo alone. These results were also shown in mouse models, showing macroscopically noticeable effects in the group treated with topical ketoconazole 2% versus the placebo group. Large, controlled studies are needed to further investigate and confirm these reports. It is unclear exactly how this antifungal shampoo helps with hair growth. Its antiinflammatory properties have been well documented. This may have to do with a reduction in Malassezia colonization of the skin. Some hypothesize that ketoconazole plays a role in local disruption of the DHT pathway. They suggest that when used in conjunction with finasteride, it may help achieve more complete reduction of DHT. This androgen-inhibiting mechanism may explain the side effect of gynecomastia seen in some patients taking oral ketoconazole. The oral administration of ketoconazole for AGA is limited by its inhibition of the biosynthesis of adrenal glucocorticoids. The drug inhibits cytochrome P-450 enzymes involved in steroid hormone biosynthesis, ultimately reducing the production of both glucocorticoids and androgenic steroids. It also has been used for hirsutism with some success.

**Nizoral shampoo**

Using the patented active ingredient ketoconazole, this topical anti-dandruff shampoo addresses the symptoms of an irritated scalp. While at first blush seemingly unrelated to the treatment of traction alopecia, Nizoral shampoo has actually been reported to slow down the effects of hair loss by inhibiting the binding of DHT to the hair follicles. Apply this treatment directly to the affected area and allow to sit for 3–5 minutes before rinsing off completely. Recommended strength is 2%, used twice weekly for the first 2–4 weeks of treatment, and every 1–2 weeks after that as a preventative measure.

**Tricomin**

Using the strength of copper peptides, Tricomin can halt or reverse the process of hair loss. This vital micro nutrient is essential to growing healthy hair, and FDA trials have shown Tricomin to be effective in improving the synthesis of collagen and mending damaged hair and skin. Tricomin is available in a variety of forms, including follicle spray, shampoo and conditioner, and recommended use depends on
the type of application. Furthermore, Tricomin can be used in conjunction with Regaine or as a more natural alternative to minoxidil-based products – while both products work to stimulate new hair growth, their methodologies differ, making them complementary treatments.

**Internal treatments for traction alopecia**

Biotin, Zinc, Vitamin C, MSM, Saw Palmetto, and Green Tea: these supplements can be considered a list of nutrients essential to growing healthy hair and maintaining a healthy scalp.

**MSM** (Methylsulfonylmethane) — One of the most important benefits of MSM is aiding the production of healthy cells. This quality aids in healing the scalp and damaged hair follicles in order to create stronger, healthier hair, and also makes for more beautiful skin and nails. 

Best taken in powder form and with the same toxicity as water, recommended dosage is 4 g per day. Dosage should be built up from 1000 mg to 4000 mg daily, and users of MSM should also make sure to drink plenty (at least 2 liters) of water a day. It should be noted that MSM can have a bitter taste and should be dissolved with a little warm water and mixed with a sweeter drink (like juice) to mask the taste. Otherwise tablets are also available. MSM should not be taken after 6 PM as it tends to have an energizing effect which can affect sleep. MSM should be taken for at least 6 months for optimal results.

**Vitamin C** — Generally taken in conjunction with MSM, Vitamin C boosts the immune section and aids MSM in expelling toxins and regenerating new cells. Take 1000 mg of Vitamin C along with your dose of MSM.

**Biotin** — Biotin aids cell regeneration by keeping hair in its growth state for longer periods of time.

**Saw Palmetto** — A natural anti-androgen, Saw Palmetto assists in inhibiting DHT production. Saw Palmetto is often a popular treatment option due to its economical cost and lack of side effects; however, Saw Palmetto is considered to be most effective when taken in higher doses (180 mg-3 g daily).

**Green Tea** — With its naturally high levels of antioxidants, green tea is considered to be an excellent supplemental treatment for hair loss given its anti-androgen and DHT inhibiting properties. Green tea is an excellent source of zinc, and can also be taken in capsule form (especially helpful given that it should be taken in larger doses to be effective).

**Zinc** – When used in combination with Vitamin B6, Zinc can reduce DHT levels and inhibit the production of sebum. While Zinc can be taken orally, it also can be applied directly to the affected area in the form of a topical cream. While these key ingredients can sometimes be found in generic skin and nail supplements, they are often in such low dosage that they fail to yield any sort of noticeable difference or positive results. In the long run, investing slightly more in quality supplements from trusted sources will lead to money saved overall and more importantly, real, noticeable results.

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**Supplements and over the counter products for hair growth**

<table>
<thead>
<tr>
<th><strong>Product</strong></th>
<th><strong>Mechanism of action</strong></th>
<th><strong>Reported efficacy</strong></th>
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<tbody>
<tr>
<td>Saw palmetto (Serenoa repens)</td>
<td>Inhibits 5-a reductase conversion of testosterone to DHT in the prostate, helpful in mild to moderate BPH symptoms but not helpful in moderate to severe BPH</td>
<td>One randomized, double-blind, placebo controlled trial demonstrated increased hair growth in 6/10 men with mild to moderate AGA</td>
</tr>
<tr>
<td>Biotin (vitamin H or B7)</td>
<td>Can help treat onychoschizia, increasing thickness of nails by 25%</td>
<td>No clinical trials showing efficacy treating hair loss; in vitro studies show no influence of biotin on cultured human follicular keratinocytes</td>
</tr>
<tr>
<td>Nioxin scalp therapy and treatments</td>
<td>Claims to “actively remove” excess sebum containing DHT, the most frequent cause of hair loss does not claim to block DHT</td>
<td>Not approved by the FDA, no clinical trials</td>
</tr>
<tr>
<td>Procerin tablets and topical serum</td>
<td>Proprietary blend of herbal, vitamin, and mineral components which “naturally block” DHT levels</td>
<td>Not approved by the FDA, nonclinical trials</td>
</tr>
<tr>
<td>Tricomin shampoos and treatments (triamino copper nutritional complex)</td>
<td>Targets delivery of copper to the base of the hair follicle</td>
<td>Ex vivo studies support the use of tripeptidecopper complexes to promote the growth of human hair follicles no clinical trials to date</td>
</tr>
<tr>
<td>Toppik (camouflage)</td>
<td>Keratin-based fibers which adhere to scalp and existing hairs; helps thicken the appearance of existing hairs and camouflage balding areas on the scalp; no claims to increase hair growth</td>
<td>Well-liked by patients for its easy application while awaiting new hair growth</td>
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Using Caffeine shampoos to treat hair loss.

Hair roots generally grow in an 8 year cycle, producing new hair during a growth phase that is repeated around a dozen times before the root dies. However a hereditary excess of testosterone weakens the hair roots in the scalp causing hair loss and premature end of the hair root. Testosterone achieves this by preventing the natural supply of an energy compound to the hair root, required to maintain the growth phase of the hair root.

A caffeine shampoo counteracts the reduction in natural energy compound at the hair root by providing active ingredient at the root thus promoting an extended growth phase. Alpecin double effect shampoo is one such caffeine shampoo that applied daily has been shown to remain on the scalp even after a short application period. This promotes the absorption of caffeine into the scalp and the hair follicle is provided with the energy needed to continue producing hair. As a result, premature hair loss is prevented, hair root activity is increased and the speed of hair growth is improved. These benefits also apply to people who suffer from traction alopecia, making Alpecin double effect shampoo a suitable shampoo for people with traction alopecia or thinning hair.

An independent German study has successfully shown that the caffeine component of the Alpecin shampoo is capable of countering the negative effects of testosterone on the male hair follicle that would normally lead to male pattern baldness. The study concluded that using a caffeine shampoo resulted in an average hair growth increase of over 45% and the life of the hair was increased by over one third.

Plantur 39 caffeine shampoo is also promoted as a hair loss remedy for women. As estrogen performs a natural protection for women's hair follicles early in their life, it is only after the menopause that an increase in testosterone can lead to hair loss for women. Daily treatment with Plantur 39 caffeine shampoo can counter act this damaging increase is testosterone and promote a longer more productive growth cycle for the hair follicle.

For men and women alike, caffeine shampoo is helping improve the condition of the scalp and encourage a healthier head of hair, thanks to daily treatments of caffeine.

Low-level light therapy

A number of products using low-energy laser light beams have been marketed for hair growth. They are available without a prescription and are usually sold directly over the Internet or through late-night infomercials. Most are packaged like a hairbrush or comb which shines red light directly on the scalp while it is used to comb through the hair. Only one such device, called the Hair Max Laser Comb (Lexington International, LLC, Boca Raton, FL), has obtained 510K FDA approval for use as a medical device. The earliest evidence that low-level light therapy (LLLT) could help with hair growth was provided by Hungarian researcher Mester in 1967. He found that by shining a low-powered ruby red laser (694nm) on the backs of shaved mice, he could increase their hair growth. This was the origin of biostimulation, using “cold laser” or “soft laser” therapy administered at lower powers of 1 to 500 milliwatts. Since then, basic research has demonstrated that LLLT can improve wound healing, reduce inflammation, and reduce the symptoms of stroke. Nonetheless, its mechanism of action is not yet known. Some have proposed that LLLT can enhance the local production of adenosine triphosphate by mitochondria. Indeed, there is evidence that it increases the activity of complexes II and IV in the mitochondrial respiratory transport chain. LLLT is discussed as a safe option for patients
unable to use any of the medications described above, either alone or as an adjunct to hair transplant surgery. We make sure to stress the lack of independent, large-scale clinical trials documenting its efficacy. Many physicians specializing in hair agree that more studies are needed to examine its role in treating hair loss.

CONCLUSION

Traction alopecia in this cohort of women demonstrated diagnostic challenges, which were resolved 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 along with a biopsy used to confirm the diagnosis. Early intervention is vital in order to reverse hair loss in TA; the eventual outcome of hair loss frequently depends on timely diagnosis combined with appropriate counseling of patients.

With all of the options for treating hair loss, it is not surprising that patients frequently feel overwhelmed and confused. As dermatologists, we can help them sort out the data and decide which options are best for them. Personal preferences may play an important role in determining the best treatment option. Minoxidil and finasteride remain our best agents in handling hair loss. If patients are willing, we encourage them to use both. We find this to be the most effective clinical practice, and it is supported by the literature. However, depending on the patient’s lifestyle or budget, it may be difficult to use both for an extended period of time. We recommend at least a 6-month overlap when transitioning from one to the other, so that the hairs that are thickened or regrown with one treatment are not abruptly lost. Even then, there is no guarantee that patients will not experience a telogen effluvium when switching from one treatment to another. It is also important to consider and allow for the differences in the treatment of male and female pattern hair loss. Most men are aware of their diagnosis and may have a notable family history. Their examination is often straightforward. However, many women have no family history, and require a considerable history and physical examination in order to diagnose female pattern hair loss.

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