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Seminars in Cutaneous Medicine and Surgery

Approach to Hair Loss in Women of Color

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Hair loss in women of color represents a unique diagnostic challenge that requires a systematic approach. In women of color, clinical examination of the hair and scalp is most helpful when performed first and used to guide subsequent history-taking to arrive at a clinical assessment. The most common hair problems in women of color are hair breakage, traction alopecia, and central centrifugal cicatricial alopecia. A careful detailed clinical examination and history will guide the clinician to appropriate counseling and management. It is important to recognize that a patient may have more than one of these 3 diagnoses and each requires separate attention. Traction alopecia is completely preventable with appropriate education of the public and medical establishment.
Semin Cutan Med Surg 28:109-114

Clinical Examination

The patient with hair loss arrives at your office and reports: "I am losing my hair, or, my hair has stopped growing, or my hair is breaking." These words can mean drastically different things to different patients. The role of the astute clinician is to unearth their true significance with a thoughtful and stepwise diagnostic approach. In women of color, this approach is aided by beginning, somewhat unconventionally, with a clinical examination of the hair and scalp. A detailed history then follows to supplement and focus the clinician's assessment and elicit further findings of interest. Although these strategies make use of the basic principles essential to any complete hair and scalp evaluation, they are also tailored to address some of the specific issues and diagnoses more prevalent in women of color.

The clinical examination begins as soon as the patient enters the room and is facilitated by a few simple measures: Take note of the patient's hair style. Have the patient take her hair down, remove any hair piece(s) or wig, remove pins or clips, undo braids, and identify the presence of hair extensions. Have the patient sit in a chair, as opposed to an examination table; this position allows a complete evaluation of the hair and scalp from above. Good lighting is essential, and ideally includes a magnifying examination light.

Once the patient is seated, the examination ensues, taking into account the following key features:

1. **Hair loss.** To what extent is there hair loss? Is it in patches or all over? Is the scalp visible? Are there bare areas and, if so, are they "bare as a baby's bottom"? What is the pattern of thinning and where on the scalp is it?
2. **Scalp.** Is scaling present? Is scaling perifollicular, in patches, or all over? Is there broken stubble? Is erythema present? If so, is it perifollicular, in patches, or all over? Are follicular ostia present, diminished, or absent? Are follicular ostia patulous or dusky?
3. **Hair breakage.** The blunt or frayed ends of breakage are best visualized with the use of a hair card, which is a piece of paper or card that provides both a white background for the examination of dark hairs and a dark background for the examination of light hairs. Using this tool, the clinician is able to determine whether the short hair is new growth or if it is broken hair. Is the hair of all lengths or is most of it short and close to the scalp? Distribution is also key. Where is the broken hair located—in areas of friction, over the frontal hairline, or over the occiput?
4. **Traction hair loss.** Is thinning present mainly at hair margins, behind the frontal hairline, or at the temporal scalp?
5. **Condition of hair.** Is the hair fragile or strong? Does it pull apart into little bits with a gentle "tug test"? Feel the hair. Does it feel silky, brittle, or coarse?

The clinician must remember that more than 1 issue can occur in the same patient. Both hair loss and breakage may be

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Authorship contributions: Dr Jennifer M. Fu and Dr Vera H. Price had full access to all content included in the manuscript and take responsibility for its integrity and accuracy. Other responsibilities and contributions include (1) manuscript concept and design (Price); (2) administrative, technical, and material support (Fu, Price); (3) drafting of manuscript (Fu, Price); (4) critical revision of manuscript (Fu, Price); and (5) manuscript supervision (Price).
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1085-5629/09/$-see front matter
doi:10.1016/j.sder.2009.04.004

present. Diagnoses may coexist—for example, trichorrhexis nodosa, traction alopecia, and cicatricial alopecia may all occur in one patient. It is critical that the clinician recognize this possibility and address each problem separately.

History

With practice, the aforementioned examination can be performed easily and efficiently. An ideal hair and scalp evaluation provides important diagnostic information as well as guidance for further questions and avenues of investigation. A thorough history then allows the clinician to hone in on a differential diagnosis. In women of color with hair loss, the history should begin with basic questions regarding symptom onset, duration, progression, distribution, and character: When did you or your hairdresser first note any hair problem or concern? What were the first symptoms? Did the hair loss begin centrally and then spread outwardly in all directions? Was there any other notable distribution to the hair loss? Have you experienced scaling, redness, crusting, itching, pain, tenderness, or burning? Is the hair loss still spreading compared with 1 year ago, or is it now stable?

Once the general nature of the problem is established, the history is then directed toward the more common scalp and hair issues found in women of color, namely hair breakage, traction alopecia, and central centrifugal cicatricial alopecia (CCCA). Because androgenetic alopecia and alopecia areata are found in women of color just as frequently as among the general population, questions designed to elicit these diagnoses must also be addressed. As guided by the most salient findings of the clinical examination, the thoughtful historian will ask questions that encompass hair care practices, pertinent family history, and the patient's own history of hair loss.

If hair breakage is present, a history of hair care practices is essential. Does the patient use a relaxer? It should be noted that a relaxer is often culturally referred to as a "Perm"; however, relaxers are products consisting of sodium or guanidine hydroxide to make curly hair straight, whereas permanent waves or perms are products that consist of thioglycolates used to make straight hair wavy or curly. Does the patient use a texturizer (a mild relaxer) or Jheri curl (a thioglycolate)? Does the patient use heat, in the form of hot combs, hot rollers, or ceramic flat irons? Does she brush frequently to "stimulate hair that won't grow long"? What kind of brush does she use? Of note, the boar bristle brush, although expensive, is the most damaging to hair. A plastic brush or comb with widely spaced teeth and smooth round tips is the least expensive and gentlest to hair. Does the patient use conditioner regularly after chemical or heat procedures? How often does she engage in any of these hair care practices?

If follicular ostia are absent or diminished, cicatricial alopecia becomes the primary consideration. In women of color, CCCA is the leading diagnosis, but other types of cicatricial alopecia, including chronic cutaneous lupus erythematosus, lichen planopilaris, and its variant, frontal fibrosing alopecia, do occur. In chronic cutaneous lupus erythematosus, erythematous scaly plaques with follicular plugging (and sometimes telangiectases, atrophy, and hypopigmentation or hyperpigmentation) will often be noted in the *center* of alopecic patch, whereas in lichen planopilaris, follicular hyperkeratosis and erythema occur at the *margin* of the alopecic patch. For frontal fibrosing alopecia, loss of eyebrows, in addition to alopecia of the frontal scalp, is characteristic and may be revealing. A positive family history, by contrast, may lend more support for CCCA, because this is the only cicatricial alopecia that may occur in other family members.

If the problem is predominantly thinning at the hair margins—on the sides of scalp, above the ears, and/or along the frontal hairline—traction alopecia should be considered. How long has this symptom been present? What is the patient's hair style now? Did she wear ponytails or braids as a child? If yes, how long did she wear her hair in this style: from approximately age 2-12 years? Is she brushing or massaging these areas to "stimulate hair growth"?

If the problem is thinning mostly behind the frontal hairline towards the vertex—think of androgenetic alopecia. The physical examination will reveal miniaturized hairs in affected areas, greater thinning over the frontal scalp compared with the occipital scalp and, in some instances, bitemporal recession. Ask the patient about a similar pattern of thinning in siblings, parents, uncles, aunts, and grandparents. In the authors; experience, it is best not to solely ask whether "anyone else in the family has hair loss?" Be specific and methodical when asking about the family history, because this will frequently yield more accurate responses. If the scalp in affected areas is "bare as a baby's bottom" with follicular ostia still present, alopecia areata is the likely diagnosis. Is there patchy hair loss in other areas, such as the eyebrows and eyelashes? Does the patient have a known thyroid problem, history of atopy? Is there a family history of alopecia areata, atopy, or autoimmune disease?

Common Diagnoses and Their Management

Hair Breakage: Acquired Proximal Trichorrhexis Nodosa

In this section, a distinctive clinical picture is presented. The hair is so fragile that it breaks proximally, giving the appearance of having been cut close to the scalp.[1] Acquired proximal trichorrhexis nodosa appears at all ages and in both sexes. There may be a family history of similar "short hair." The distinctive short hair is present in large areas, such as the occipital scalp, or frontal scalp, but not the entire scalp (Fig. 1). The patient may cut the unaffected hair to approximate the length of the short broken hair (Fig. 2). There is no alopecia or hair loss associated with the hair breakage (Figs. 1 and 2).

The onset of hair breakage may occur after years of straightening with chemicals and/or heat without a problem. Sometimes, excessive brushing with a stiff brush precedes the breakage. In some patients, there is no clear history of chemical, thermal, or mechanical injury. It is important to stress that breakage does not necessarily mean that something was improperly done; rather, the effects of these practices are *additive* and, depending on the intrinsic, genetic aspects of

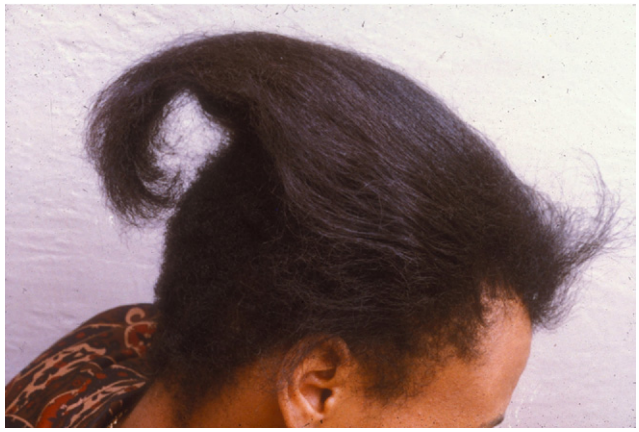

Figure 1 Proximal trichorrhexis nodosa: hair breakage over occiput.

Figure 3 "Tug test" to demonstrate hair fragility. A cluster of hair is grasped at the proximal end while the distal ends are gently tugged. Fragile hair breaks into small bits which show characteristic fractures in a hair mount.

the hair, the cumulative result is breakage. Some hair is very resistant and withstands these practices over a lifetime without breaking; other hair is weak and easily broken. Family members may have had a similar problem.

A "tug test" (Fig. 3), performed by holding approximately 10 to 20 hairs at their base with the index finger and thumb of one hand and "tugging" at their distal ends with the index finger and thumb of the other hand, demonstrates hair fragility to the patient. A hair mount is also invaluable and is made by placing the shortest broken bits of hair shaft on a glass slide, adding a drop of mounting medium (such as Permount) and a coverslip, and examining under low power with a light microscope. Trichorrhexis nodosa fractures and longitudinal splitting of the hair shaft should be evident (Fig. 4). Very curly hair also forms spontaneous knots (trichonodosis); these knots contribute to sites of breakage and may also be observed. We have found it instructive to bring the patient to the microscope. The patient will be impressed with this graphic demonstration and motivated to follow your recommendations:

1. Increase the interval between use of chemical relaxers and the use of heat. Use chemical relaxers only every 8-12 weeks. Daily blow-drying and brushing for 15 minutes will contribute to hair breakage; of note, the culprit is the 15 minutes of brushing, not the blow-drying, which does not directly contact the hair. Heat that is applied directly on the hair, as with curling irons, hot combs, hot rollers, or ceramic flat irons may be problematic. If pertinent, the clinician should correct the mistaken impression that ceramic flat irons, a relatively recent method of straightening hair with heat, are more gentle.[2] Hair that has been colored or bleached is more susceptible to breakage with heat (Fig. 5).
2. Handle hair as gently and as little as possible. Use a gentle brush (with widely spaced plastic bristles and rounded tips) and/or a gentle wide-toothed plastic comb to put hair into place. Brushing should not be excessive and should not be used for purposes of "stimulating" hair growth.
3. Avoid massaging or rubbing the hair or scalp. If the patient's scalp is itchy because of mild seborrheic dermatitis, an antidandruff shampoo may be of benefit; if the pruritus stems from another process, this should be

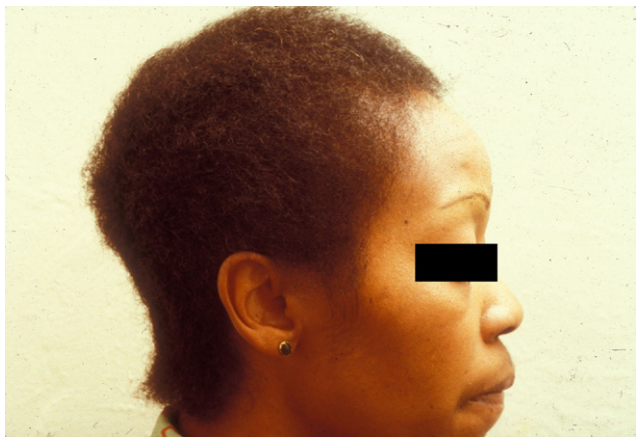

Figure 2 Proximal trichorrhexis nodosa: hair breakage over frontal scalp with remaining hair cut to approximate short length of broken frontal hair.

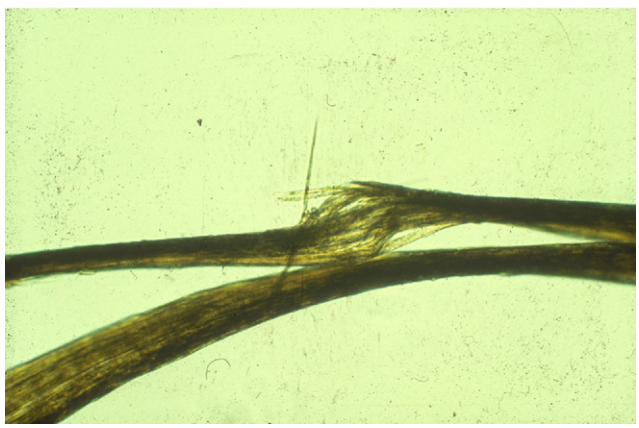

Figure 4 Hair mount showing trichorrhexis nodosa fracture and longitudinal splitting of hair shaft.

addressed with appropriate therapy. Instruct the patient not to use a comb or fingernails to scratch as this causes more breakage.
4. Use a regular conditioner.
5. A hair piece is a good way to give hair a rest from chemicals and heat.

Although the diagnosis and management of acquired proximal trichorrhexis nodosa seem straightforward, several questions regarding pathogenesis remain. It is not known, for instance, why the breakage continues for so long. The hair breakage associated with acquired proximal trichorrhexis nodosa usually continues for 2 to 4 years, even after all hair straightening procedures and vigorous grooming practices are discontinued. Possibly all the affected hair follicles must be replaced with new anagen follicles before the breakage ceases. It has also been observed that some women are more prone to developing acquired proximal trichorrhexis nodosa, suggesting that there may be an underlying genetic susceptibility in those who are diagnosed with this type of hair breakage. There may be a heritage of African ancestors with a predisposition to either long or short hair, with the short-haired ancestors conferring a predisposition to fragile hair.

Figure 5 Heat-induced hair breakage caused by ceramic flat irons. Note that the colored hair is more susceptible to heat-induced breakage. Reprinted with permission.[2]

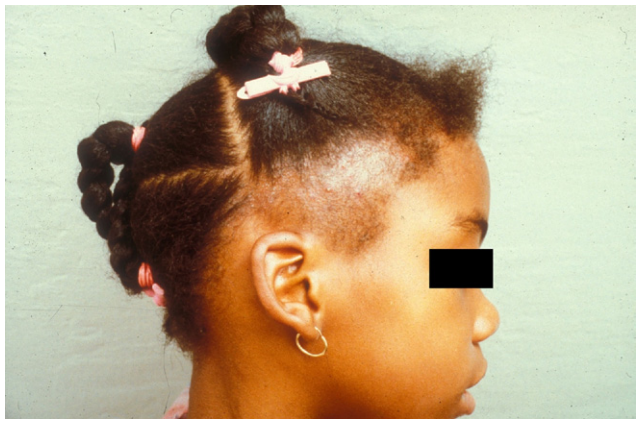

Figure 6 Traction alopecia in a young child. The traction on the hair in this hair style caused traction alopecia along the temporal hair margins. Inflammatory papules can be seen in the alopecic area. This hair loss is reversible if the traction is loosened, and hair brushing and any pulling on the hair is avoided.

Traction Alopecia

Traction alopecia is preventable and urgently calls for education and public awareness to erase this unnecessary hair problem. It frequently represents years of hair stylizing that puts traction on the hair, and causes thinning at the temporal and frontal hair margins. A favorite hair style for African-American girls is tight neat braids that are worn from about the age of 2 until the ages of 10-12 (Fig. 6). The traction often causes an associated folliculitis, which presents as asymptomatic perifollicular pustules. If traction alopecia is recognized in childhood or adolescence, and the hair style is loosened and brushing "to stimulate new growth" is stopped, along with all practices that put tension on the hair, there is usually full recovery and regrowth within a few months. However, if the patient presents as an adult (Fig. 7), and she states that the thinning at her temporal and frontal margins

Figure 7 Traction alopecia, present since adolescence, and hair breakage in an adult. This traction alopecia is no longer reversible. Note the rim of terminal hairs marking the original frontal and temporal hair lines.

Table 1 Working Classification of Primary Cicatricial Alopecias*

Lymphocytic
Lichen planopilaris
Frontal fibrosing alopecia
Graham Little syndrome
Classic pseudopelade (Brocq)
Central centrifugal cicatricial alopecia
Chronic cutaneous lupus erythematosus
Keratosis follicularis spinulosa decalvans
Neutrophilic
Folliculitis decalvans
Tufted folliculitis
Dissecting cellulitis/folliculitis (perifolliculitis abscedens et suffodiens)
Mixed
Folliculitis (acne) keloidalis
Erosive pustular dermatosis
Nonspecific (end-stage)

In this classification, classic pseudopelade of Brocq is defined as clinically discrete, smooth, flesh-toned areas of alopecia without follicular hyperkeratosis or perifollicular inflammation. Central centrifugal alopecia is defined as hair loss starting in the central scalp and progressing centrifugally. This entity has previously been referred to by other terms (follicular degeneration syndrome, pseudopelade in African-American patients, and central elliptical alopecia in Caucasian patients), but it was suggested that "central centrifugal cicatricial alopecia" is a more descriptive term that effectively embraces all prior entities. Nonspecific or end-stage cicatricial alopecia is defined as an idiopathic scarring alopecia with inconclusive clinical and histopathologic findings. This category often includes the end-stage of a variety of inflammatory cicatricial alopecias, such as lichen planopilaris and folliculitis decalvans.

*Reprinted with permission.[5,6]

has been present since her adolescence, the hair follicles have likely undergone years of perifollicular inflammation with the subsequent formation of fibrous scar tissue. The hair loss at that point is permanent. If areas of involvement are focal, a surgical treatment approach to long-lasting, permanent traction alopecia may be considered.[3]

Sometimes, adults present with a relatively short history of traction alopecia. In such cases, the clinician should inquire whether the patient wears rollers overnight and again about hair stylizing. The main differential diagnosis of acute onset traction alopecia is alopecia areata. In traction alopecia, residual terminal hairs will be observed within the affected area and will also outline the outer margins of the temporal and frontal hairlines (Fig. 7). In contrast, the affected area in alopecia areata may be entirely bare; no terminal hairs outline the outer margins of the hairlines. Despite these clinical differences, a biopsy is sometimes needed to differentiate the 2 conditions. Alopecia areata, when not extensive, responds well to intralesional injections of triamcinolone acetonide. Of interest, intralesional triamcinolone acetonide injections may also benefit acute traction alopecia by reducing localized perifollicular inflammation.

In summary, traction alopecia is reversible if recognized early and all traction on the hair is removed in childhood or adolescence. Vigorous brushing does not stimulate new growth; it only causes hair breakage. In adults, if traction alopecia has been present for many years, it is probably permanent. Hair breakage, which is often coexistent, must be addressed separately. Traction alopecia presenting as an acute problem must be differentiated from alopecia areata. Both conditions may benefit from intralesional injection of corticosteroids. Traction alopecia is a preventable problem, and as such, it is important to increase public awareness of the simple measures that prevent it.

CCCA

Cicatricial alopecia (scarring alopecia) refers to a group of disorders that destroy the hair follicle and cause permanent hair loss. The cicatricial alopecias may be primary or secondary. The secondary group results in nonspecific follicular destruction from a variety of insults, including thermal burns, metastatic cancer, infection, or radiation. In the primary group, a targeted folliculocentric inflammatory attack replaces the follicle with scar tissue and leads to progressive, permanent hair loss.[4,5] A working classification of the primary cicatricial alopecias is based on the predominant cellular infiltrate, whether lymphocytic, neutrophilic or mixed (Table 1).[5,6] The primary cicatricial alopecias present several clinical variants that usually are readily distinguishable from one another clinically. In contrast, the histopathologic features do not distinguish the clinical variants and can only separate the predominantly lymphocytic group from the predominantly neutrophilic group.[4]

CCCA is a primary cicatricial alopecia with a predominantly lymphocytic inflammation. It occurs typically in women of African ancestry, usually between the ages of 25-65 years. The patients, who are otherwise in good health, first note a small area of thinning over the central scalp. This gradually enlarges and spreads centrifugally in a symmetric pattern to affect a large area over the top and sides of the scalp with a few residual terminal hairs in the bare areas (Fig. 8). The affected scalp shows a loss of follicular ostia, with residual hairless ostia appearing patulous and dusky. In most cases, there are only mild associated symptoms, such as episodic itching or tenderness.

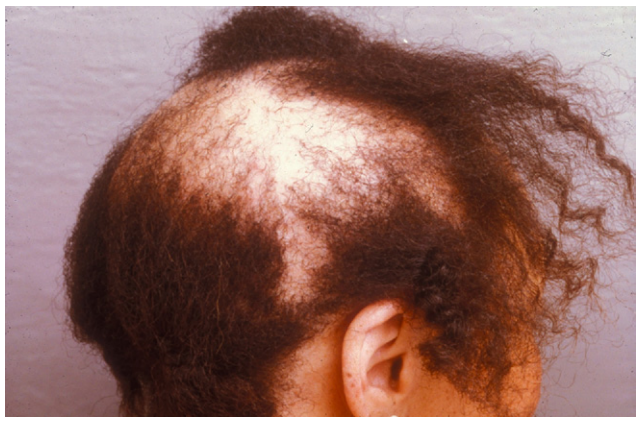

Figure 8 Central centrifugal cicatricial alopecia.

The cause of CCCA is unknown, although a combination of genetic factors and inflammation appear to play a role. Women with CCCA may have female relatives with a similar problem, which makes CCCA the only cicatricial alopecia in which other family members may be similarly affected.

Hair breakage and traction alopecia are 2 hair problems not infrequently present in some women with CCCA. Although both of these disorders can be attributed to hair grooming and stylizing practices, at present the relationship of these hair grooming and stylizing practices to CCCA is still debated and is being determined. It is worthwhile to emphasize that the histology of CCCA is indistinguishable from the histology of other types of lymphocyte-mediated cicatricial alopecia.

When the diagnosis of CCCA is suspected from the clinical appearance of the scalp, a scalp biopsy will determine whether the disease is active and requires treatment to try to arrest the process or whether the process is largely completed and will no longer benefit from therapy. The type, extent, and location of the inflammatory infiltrate; the presence or absence of sebaceous glands; the degree of scarred fibrosis; and the number of remaining anagen follicles, all help in this determination. In CCCA, despite impressive loss of hair over a large scalp area, the symptoms and signs are often minimal and the main indicator of activity and the need for treatment is the patient's observation that the hair loss is still spreading.

It is important to explain to the patient that the goals of treatment are to halt spread and to relieve symptoms and signs. Hair regrowth is not possible after the hair follicles are replaced by scar tissue. Therefore, early recognition and early initiation of treatment are important. There is no single treatment for CCCA, and clinicians experienced in managing CCCA may treat it somewhat differently.[7] The following treatment guidelines reflect the practice of the authors.

Active spreading disease is an indication for oral treatment, including oral antibiotics with anti-inflammatory effects, such as doxycycline, 100 mg twice daily, or antimalarials, which are antilymphocytic and steroid-sparing, such as hydroxychloroquine, 200 mg twice daily.[5] Before administering hydroxychloroquine, a baseline retinal examination, complete blood count, G6PD (glucose 6-phosphate dehydrogenase), and liver function tests are documented. The blood tests usually are not repeated during the course of treatment; the ophthalmologist usually requests a follow-up examination in 6-12 months. We have not had any occurrence of retinopathy with hydroxychloroquine, in keeping with other centers.[8] Both doxycycline and hydroxychloroquine have been well tolerated except for occasional gastrointestinal intolerance. Patients are clinically assessed every 3 months. We have occasionally needed to use mycophenolate mofetil or cyclosporine for recalcitrant, active, and spreading disease.[5]

Topical therapies for relief of symptoms or if the biopsy shows dense perifollicular inflammation include high-potency topical corticosteroids, topical tacrolimus or pimecrolimus, and intralesional injections of triamcinolone acetonide, 10 mg mL^{-1}. Derma-Smoothe/FS scalp oil (Hill Dermaceuticals, Sanford, FL) is helpful for the treatment of intense scaling and itching. Topical minoxidil has been used to prolong the anagen phase of any residual follicles that might still benefit from this stimulation, and for androgenetic alopecia if coexistent. One last comment, if a patient suddenly develops intense pruritus in the course of her treatment for CCCA, ask if she has grandchildren. The cause might be *Tinea capitis*, not an exacerbation of CCCA![9]

Conclusions

A systematic approach in assessing hair loss in women of color will demonstrate the 3 most common problems, hair breakage, traction alopecia, and CCCA. The clinician should be alert to the possibility that more than one of these conditions frequently occurs in the same patient, and each must be addressed separately.

Acknowledgments

We are grateful to Dr Paradi Mirmirani and Dr Charles Chiang for their contribution to the manuscript. We would also like to thank Marilyn Hadden for her administrative assistance.

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