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Review

Trichoscopy – a valuable tool for identifying conditions mimicking androgenetic alopecia

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Abstract

Androgenetic alopecia (AGA) is the most prevalent type of hair loss in women and men. Recently, a European consensus group published guidelines for the diagnostic evaluation of AGA in men, women, and adolescents. This S1 guideline presents expert opinion-based recommendations for gender-dependent steps in the diagnostic procedure, which can easily be implemented in the daily clinical routine. For diagnosing AGA, detailed anamnesis and objective learning are not enough because there are several conditions mimicking this disease. Trichoscopy can be considered an important, non-invasive tool for diagnosing hair and scalp disorders that may have similar clinical signs to AGA.

Introduction

Androgenetic alopecia (AGA) is the most prevalent type of hair loss in women and men.[1,2] It is characterized by progressive hair thinning and has distinctive patterns of hair loss in women versus men, but in both genders, the central scalp is most severely affected.[3]

For diagnosing AGA, detailed anamnesis and objective learning are not enough because there are several conditions mimicking this disease.

Recently, a European consensus group published guidelines for the diagnostic evaluation of AGA in men, women, and adolescents. This S1 guideline presents expert opinion-based recommendations for gender-dependent steps in the diagnostic procedure, which can easily be implemented in the daily clinical routine.[4] The important tool for formulating a diagnosis of hair and scalp disorders is trichoscopy, or hair and scalp dermoscopy.[5–9]

Mimickers of AGA can be distinguished in two major groups on the basis of the hair loss distributions:

Diffuse hair loss

Telogen effluvium (TE)

Telogen effluvium is acute or chronic, non-inflammatory, diffuse hair loss that may be most difficult to distinguish from female pattern hair loss (FPHL),[10] which is characterized by a diffuse thinning of the centro-parietal area with preservation of the frontal hairline.[1] The clinical history and strongly positive pull-test are often valuable for identifying this diagnosis. An acute febrile illness, severe psychological trauma, significant weight loss, childbirth, certain medications, major surgery, and nutritional deficiency may precipitate an episode of TE that usually begins 3 months after the insult.[11,12]

On trichoscopy, TE is mostly a diagnosis of exclusion.[13] According to the study by Rakowska et al., trichoscopic

examination may be sufficient for making a differential diagnosis between female AGA and chronic TE.[14] The trichoscopic features that we see in TE are not specific and are detected in many other types of alopecia. The main difference between AGA and TE is the location of trichoscopic features on the whole scalp and not only on the androgen-dependent area.[15] We have to take into account the common fact of the coexistence of the above-described two diseases. In these cases, detection of AGA is possible by well-defined trichoscopic features of the disease, but for identification of the early stage of AGA histomorphology study is needed.[11,16,17] Diagnosing may be challenging because of the bitemporal recession that may be seen in patients with TE,[15,18–20] which makes it similar to male AGA.

Trichoscopic features that we may see in TE are decreased hair density with empty hair follicles, predominance of follicular units with only one hair, perifollicular discoloration, upright regrowing hairs (tip-pointed regrowing hairs), and lack of features typical of other diseases.[13,15]

Lichen planopilaris (LPP) and fibrosing alopecia in pattern distribution (FAPD)

Lichen planopilaris (LPP) is the most common cause of cicatricial alopecia in adults.[21,22] When there is the classic lesion of LPP (whitish atrophic or scarring patches with complete loss of follicular orifices),[23] it is easier to make a differential diagnosis with AGA. However, as the central scalp is a common target and in the early stages of the disease, little to no associated alopecia may be found,[24] LPP can mimic AGA (Figure 1), and the disturbing sensations on the scalp may be determined to be scalp trichodynia that is often seen in AGA.[25] One of the common signs of LPP, which ranges from mild to severe perifollicular scaling, can be dermatologically evaluated as seborrheic dermatitis, which also often coexists with AGA.[26]

Starace et al. described 40 patients affected by diffuse hair thinning associated with a long-lasting history of pruritus and erythema of the scalp and a histopathologic diagnosis of LPP. A new variant of diffuse LPP, named "lichen planopilaris diffuse pattern," was described in 50% of patients.[27]

Trichoscopy of LPP may reveal: perifollicular erythema and scaling; scales entangling hair shafts up to 2–3 mm above the scalp surface in a tubular manner; hair casts; elongated linear blood vessels; violaceus areas; dystrophic hairs (pili torti); rarely broken hairs and tufted hairs. Along with the progression of the disease with trichoscopy, we may find the absence of follicular openings and fibrotic white dots, white areas, and milky red areas. Vellus hairs are usually absent.[8,26–30] Trichoscopy features are useful not only for diagnosing early stages of the disease but are also helpful for calculating the LPP activity index.[22]

In medical literature, we come across some cases of LPP in children when the final diagnosis is made only according to histomorphology.[31] In patients suspected of having lichen planopilaris, the biopsy site should contain hairs with perifollicular scaling/casts or small tufts surrounded by casts.[6]

Fibrosing alopecia in pattern distribution (FAPD) was described in 2000 by Zinkernagel and Trüeb as a new form of cicatricial alopecia. FAPD is a diffuse, non-patchy cicatricial alopecia limited to the area of androgenetic hair loss[24] (Figure 2).

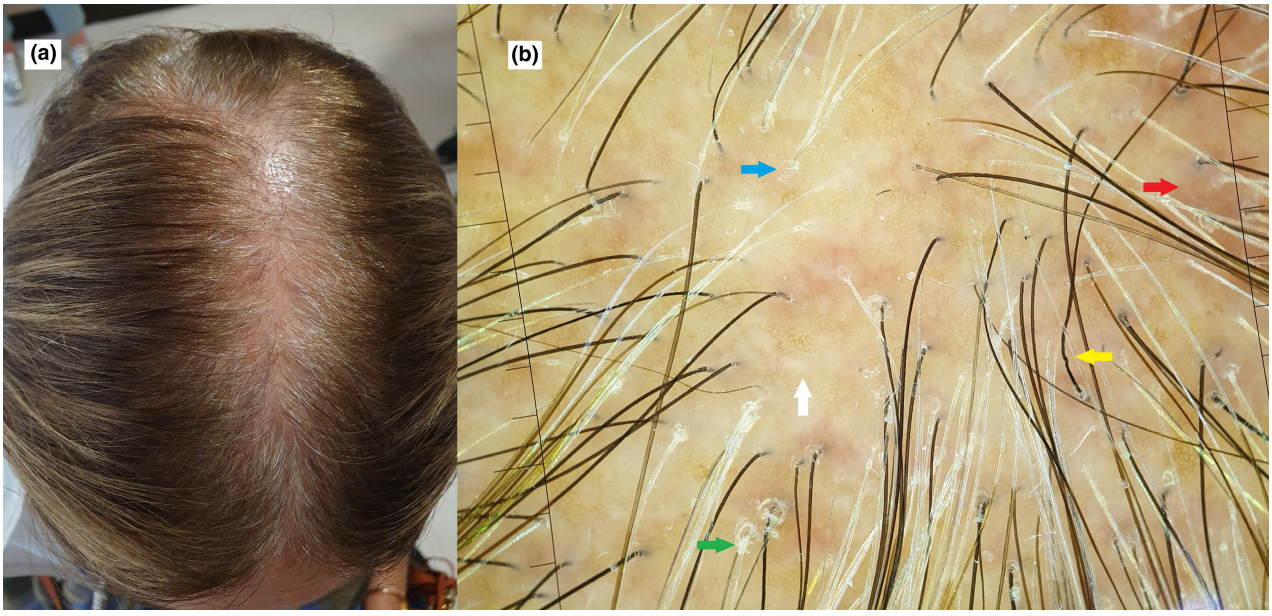

Figure 1 Clinical (1a) and trichoscopic (1b) images of 35-year-old women with LPP. Trichoscopy shows perifollicular scaling (blue arrow), hair casts (green arrow), violaceus areas (red arrow), dystrophic hairs (yellow arrow), and fibrotic white dots (white arrow)

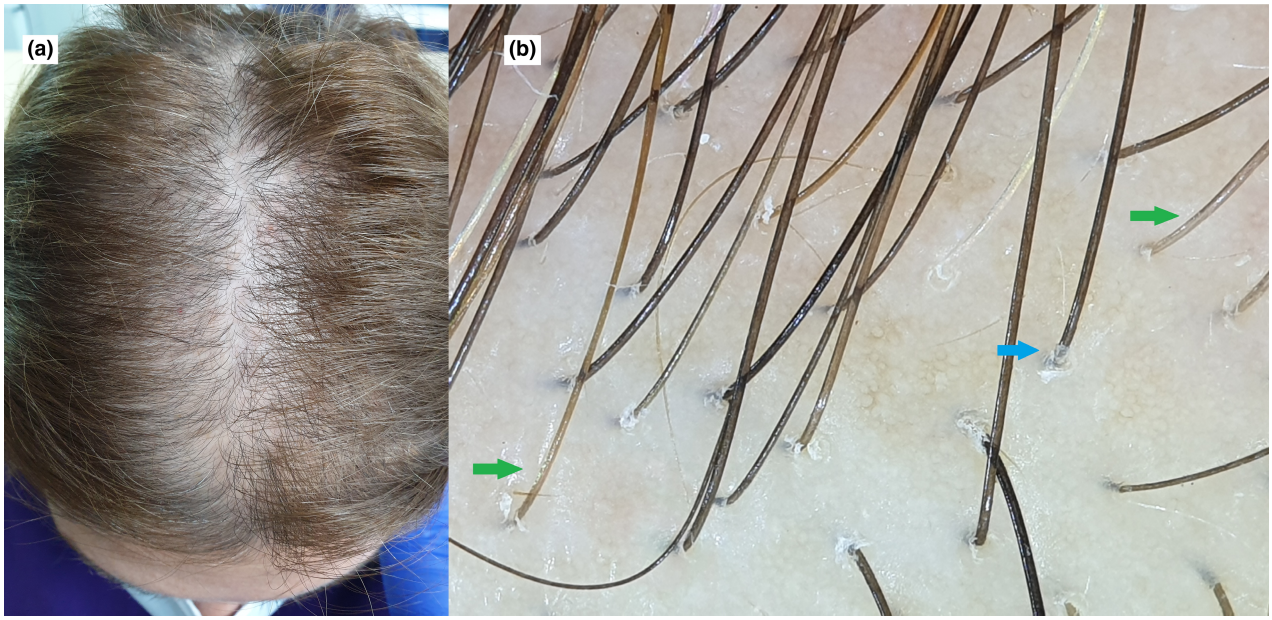

Figure 2 Clinical (2a) and trichoscopic (2b) images of 42-year-old women with FAPD. Trichoscopy shows hair diameter diversity (hairs of different thickness), perifollicular scaling (blue arrow), and the predominance of single hair follicles (green arrows)

Patients usually complain of a long-lasting history of hair loss variably associated with scalp dysesthesia.[32]

Trichoscopy of FAPD shares similar features characteristic of AGA: hair diameter diversity, peripilar sign, and yellow dots, but the predominance of single hair follicles, perifollicular erythema, perifollicular scaling, and perifollicular white halo indicate cicatricial alopecia.[33] Partial or total loss of the follicular openings may also be seen.[34,35]

Individual or compound hairs with peripilar concentric or tubular casts are the optimal site for obtaining the scalp biopsy in FAPD.[36]

In 2005, Olsen coined the term cicatricial pattern hair loss to describe a form of cicatricial alopecia in a case of FPHL, lacking the perifollicular erythema and follicular hyperkeratosis seen in FAPD. Typically, it affects women over 40 years, and the specific trichoscopic sign is the presence of small "pencil-eraser-sized" areas of focal atrichia.[37]

Central centrifugal cicatricial alopecia (CCCA)

Central centrifugal cicatricial alopecia (CCCA) is a common condition that mostly affects women of African descent and may occur in families.[28,38] In contrast to the pattern of hair loss, the disease clinically presents as patches of permanent hair loss on the vertex or crown of the scalp and spreads centrifugally.[24,39,40]

The aim of the study by Miteva and Tosti was to establish the spectrum of dermoscopic features and frequency in CCCA. Results showed peripilar white-grey halo around the emergence of hairs in 94% of patients and was highly specific and sensitive for CCCA in all clinical stages.[41] Trichoscopy of CCCA also shows a preserved honeycomb pattern and an irregular distribution of pinpoint white dots. Irregular white patches corresponding to follicular scarring are scattered between the dots. Also, broken hairs, black dots, asterisk-like brown blotches, and a dark peripilar halo may be seen. Erythema is often detected, but vascular pattern is not visible due to the existence of pigmentation.[26,28] The key feature to differentiate AGA from CCCA is the rapid development and localization of trichoscopic features to the vertex, with sparing of the mid-frontal scalp.[40]

The best biopsy site in CCCA is white/grey peripilar halos and/or broken hairs.[6,42]

Patterned hair loss

Hairline maturation (HLM)

Rassman et al.[43] observed the process of how the hairline develops from childhood to adulthood in men and women. They conclude that the slight recession of the hairline is a normal, hormonally mediated occurrence that typically begins in early adulthood. The limited extent of hairline recession contrasts with the more extensive recession and vertex involvement that occur in AGA. One can figure out if a male has hairline maturation (HLM) or AGA by performing the forehead wrinkle test. If one wrinkles their forehead upward, the normal hairline in young males is attached to the upper wrinkles. HLM moves it back further (about one fingerbreadth), and male balding moves it even further. The changes for HLM are about two finger breadths in the temple area, and anything more is likely suggestive of AGA. There is limited information about the trichoscopic features of HLM in medical literature.

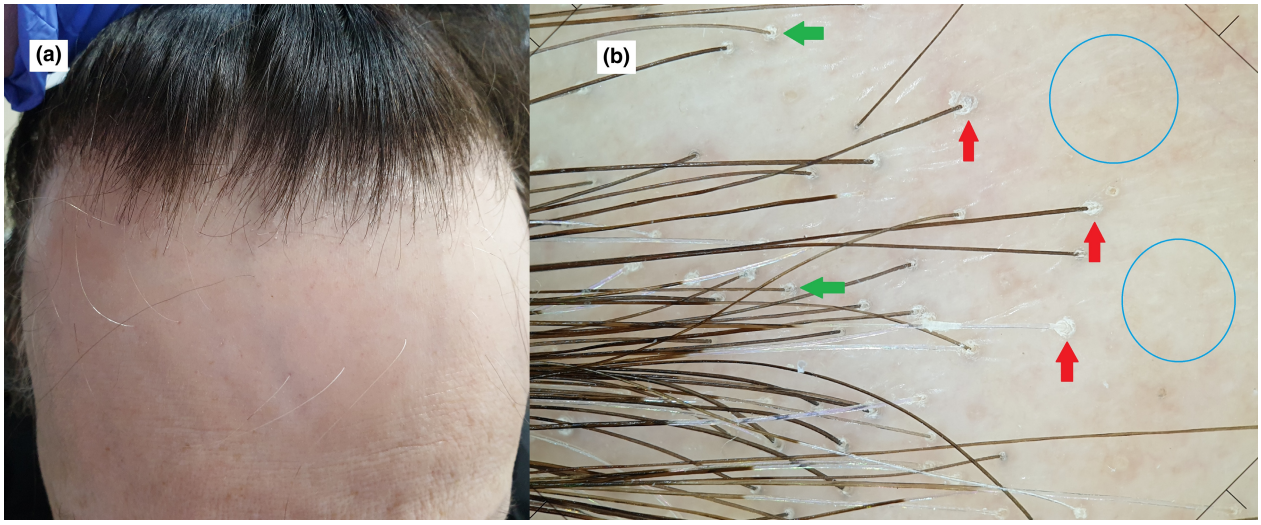

Figure 3 Clinical (3a) and trichoscopic (3b) images of 48-year-old women with FFA. Trichoscopy of the frontotemporal area shows a lack of follicular openings (blue rings), an ivory-colored background (blue rings), perifollicular scaling (green arrows), lonely hairs (red arrows), and an absence of vellus hairs (normally, the frontal hair-bearing margin must contain vellus hair)

Frontal fibrosing alopecia (FFA)

Frontal fibrosing alopecia (FFA) is a variable progressive cicatricial alopecia that mostly affects women in the postmenopause. It commonly presents with symmetric, marginal alopecia along the frontal and frontotemporal hairlines and loss of eyebrows.[18,20,24,26,44,45]

Due to frontotemporal onset with slowly progressive bitemporal recession, FFA becomes similar to male pattern hair loss (MPHL)[34] (Figure 3), which is presented with a recession of the frontal hairline, mainly on a frontotemporal area of the scalp in a triangular pattern bilaterally, later followed by a vertex thinning.[1] Unlike MPHL, trichoscopy of FFA may reveal minor perifollicular scaling and erythema, a lack of follicular openings, an ivory-colored background, and pili torti on the hair-bearing edge.[26,28,44] Lonely hairs and an absence of vellus hairs have been reported as potential clues for FFA.[18]

The International Dermoscopy Society members described 188 cases of FFA, demonstrating a predominant female population (98.4%), and in 71.8% of the cases, the clinical presentation and the trichoscopic findings were decisive for the diagnosis without the necessity of a biopsy.[46]

In addition, in FFA, one more benefit of trichoscopy is observing not uncommon types of hair loss of eyebrows and peripheral body hair. Trichoscopy of eyebrows shows regularly distributed red or gray dots,[29,44] dystrophic hairs, whitish areas with the absence of follicular openings, and eyebrow regrowth in distinct directions.[47]

The role of trichoscopy is increasing in making differential diagnosis between LPP and FFA because of the difficulty of finding histologic clear-up between these diseases.[48–50]

The best biopsy site in FFA is terminal hair with concentric scaling.[6]

Traction alopecia (TA)

Traction alopecia (TA) is a result of prolonged or repetitive tension on the hair roots and occurs in individuals with specific hairstyles or occupational uniforms.[18,51] The location of hair loss correlates with the site of traction. The clinical presentation and a thorough history are keys to diagnosis, but as the frontotemporal scalp is the most commonly affected area at the beginning, this condition can mimic AGA (Figure 4). TA is a diagnostic challenge when the external factor is not suspected or admitted. If the cause of TA persists for a long period, permanent scarring alopecia may occur[18,51] and in those cases, we need to exclude primary cicatricial alopecias.

The trichoscopic findings vary by the duration of traction.[52] Trichoscopy shows features characteristic of AGA: reductions in hair density, hair diameter diversity, empty hair follicles, hair thinning, and persistence of vellus hairs. However, unlike AGA, trichoscopy reveals perifollicular erythema and atypical red vessels and hair casts. Black dots, yellow dots, broken hairs, commas, and coiled hairs may also be seen.[18,28,52–55]

Mixed hair loss

There are several hair conditions that may have both diffuse or patterned distribution.

Trichotillomania (TTM)

Trichotillomania (TTM) is an obsessive-compulsive type of disorder characterized by senseless and undesirable hair-pulling behavior. The condition may be episodic but is usually chronic and difficult to treat.[56] TTM may involve any area of the body but more often the scalp.[10]

A thorough history, clinical appearance, and trichoscopy are the keys to diagnosis.[57] Trichoscopy reveals abnormalities

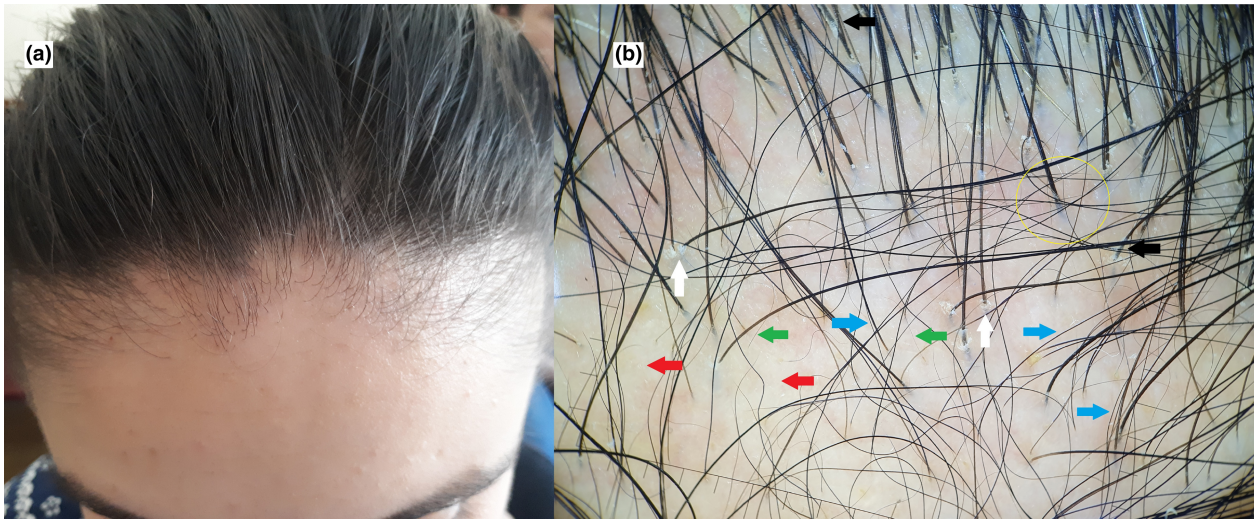

Figure 4 Clinical (4a) and trichoscopic (4b) images of 22-year-old women with TA. Trichoscopy of the frontotemporal area shows a reduction in hair density (large difference between the frontal hair bearing margin and central scalp), hair thinning (blue arrows), persistence of vellus hairs (green arrows), empty hair follicles (red arrows), perifollicular erythema (yellow ring), perifollicular scaling (white arrows), and hair casts (black arrows)

resulting from the stretching and fracture of hair shafts.[10] Trichoscopic features include: hairs broken at different lengths; coiled hairs; hook hairs; trichoptilosis; flame hairs; tulip hairs; V-signs; and hair powder.[53,58,59]

In most cases, TTM must be differentiated from patchy alopecia areata because on the one hand, clinically and trichoscopically, they look similar, and on the other hand, both diseases may coexist. However, there are some cases when clinically, TTM might mimic female (Figure 5) or male (Figure 6) AGA, and trichoscopy has the main role in the differentiation of these conditions.

Alopecia areata (AA)

Alopecia areata is a common, inflammatory, non-scarring type of hair loss. Significant variations in the clinical presentation of AA have been observed, ranging from small, well-circumscribed

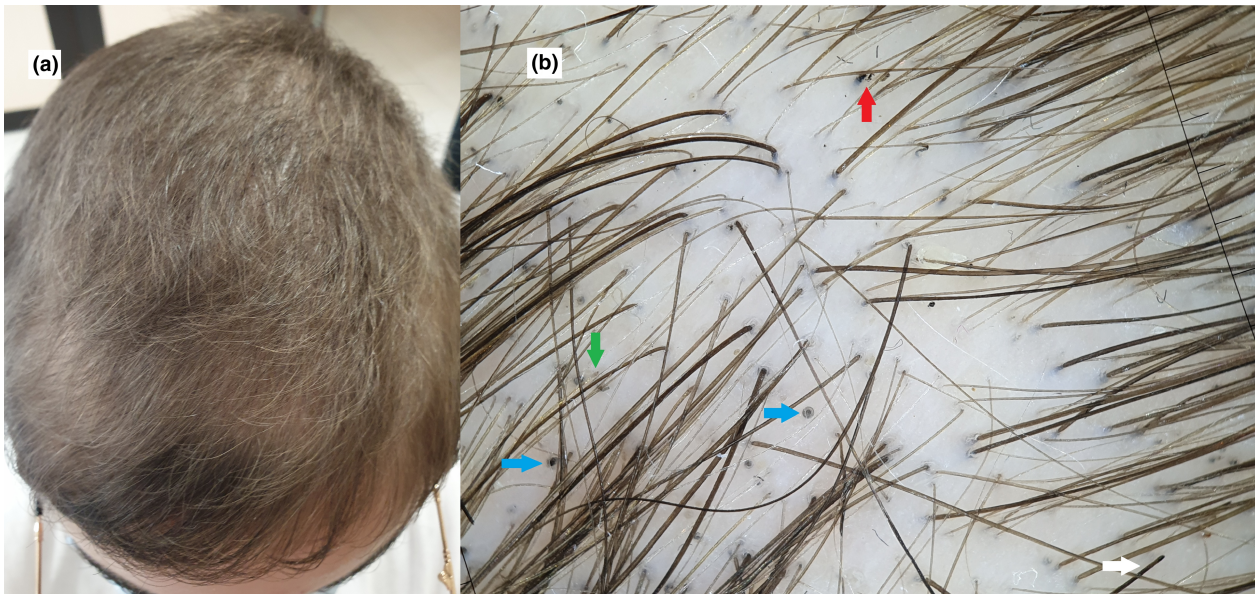

Figure 5 Clinical (5a) and trichoscopic (5b) images of a 16-year-old girl with TTM. Trichoscopy shows broken hairs at different lengths (blue arrows), hair powder (green arrow), flame (red arrow), and tulip hairs (white arrow)

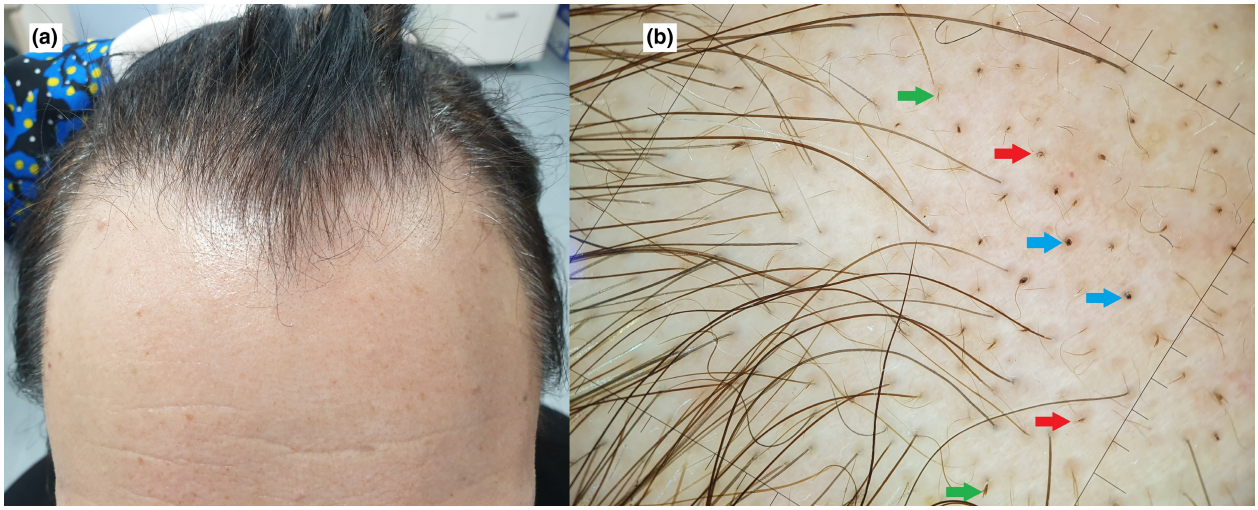

Figure 6 Clinical (6a) and trichoscopic (6b) images of 54-year-old women with TTM. Trichoscopy of the frontotemporal area shows broken hairs (blue arrows), V-sign hairs (green arrows), and trichoptilosis (red arrows)

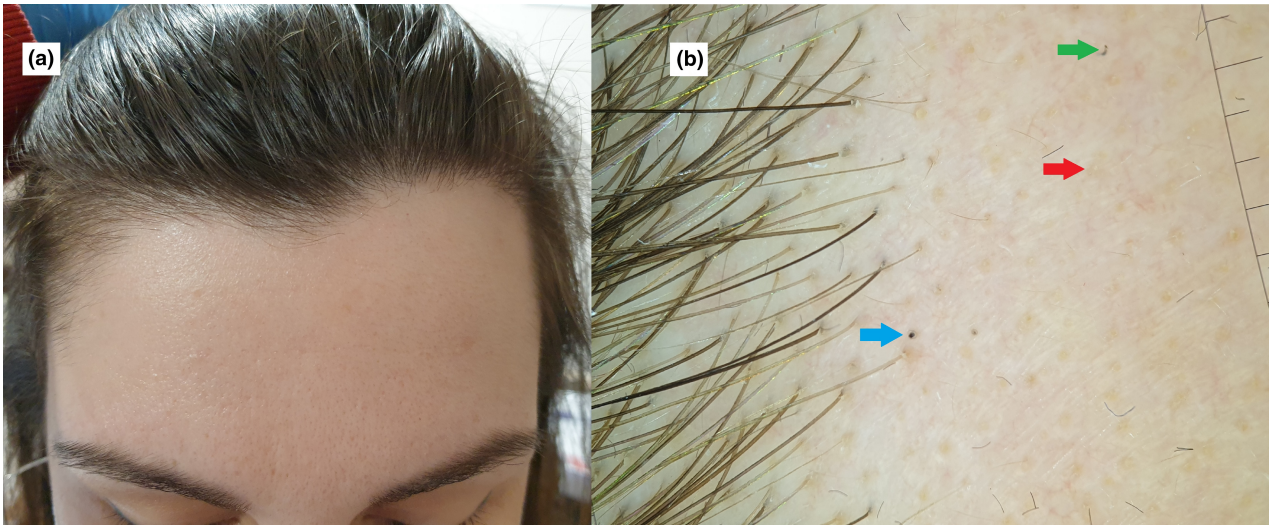

Figure 7 Clinical (7a) and trichoscopic (7b) images of 33-year-old women with alopecia areata. Trichoscopy of the left frontotemporal area shows black dots (blue arrow), broken hairs (green arrow), and yellow dots (red arrow)

patches of hair loss to a complete absence of body and scalp hair.[60] It is easy to differentiate patchy AA from AGA. However, there are clinical variants of AA: diffuse alopecia areata (DAA) and alopecia areata incognita (AAI). They are presented as diffuse hair loss and reductions in hair density.[61] AAI and DAA may seem similar to AGA. For these two conditions, clinical and trichoscopic features are decisive in making a diagnosis and also in selecting the best site for biopsy.[62]

Alessandrini et al.[62] described the trichoscopic features of AAI and DAA according to a 5-year study. The most frequent pattern was empty yellow dots, yellow dots with vellus hairs, and small hair in regrowth, but the presence of pigtail hair was found almost exclusively in those with AAI. In cases of DAA, the finding of dystrophic hairs and black dots was more frequent.

Miteva et al.[63] described 46 cases with AAI to identify its most common histopathologic features and provide pathological guidelines for the diagnosis. This study showed that in AAI, diffuse alopecia with reduced hair density may be more pronounced on the androgen-dependent scalp. Trichoscopy showed yellow dots and short, regrowing hairs. They mentioned differences between AAI and DAA, as in that entity, clinical and trichoscopic examination shows exclamation mark hairs and dystrophic hairs.

Meah et al.[64] described five cases of bitemporal alopecia areata, with involvement of the frontal hairline. The purpose of this report was to highlight the distinct presentation of AA and

recognize its clinical mimickers. They conclude that in those cases, it is important to exclude AGA and chronic TE, which can be done on history, clinical examination, and trichoscopy, but very occasionally a scalp biopsy may be indicated (Figure 7).

Starace et al.[65] described 11 cases of long-standing, isolated patchy AA on the frontoparietal hairline. Trichoscopic features were mainly yellow dots, short regrowing hairs, vellus, circle hairs, broken hairs, and black dots. Exclamation mark hairs were never detected.

Discussion

The majority of the population identifies hair thinning with the loss of hair shafts. Due to the fact that AGA does not cause intense and rapid hair loss, patients do not seek medical consultation at the early stage of the disease, which usually causes late diagnosis and treatment. Frequently, the number of consultations increases when the hair loss is speedy and the thickness of the hair changes dramatically, or when AGA coexists with TE or other types of hair loss.

When we are talking about the negative influence of the disease, it is important to underline that in men, the problem is not perceived as desperately as in women. AGA often has effects on the lives of female patients, causes a worsening of quality of life, and may be accompanied by hormonal disbalance and other disturbing problems. The disease in male patients with a disposition toward total baldness causes a visual defect and a lack of the function of protecting the scalp from the chronic effects of sunlight, heat, and cold.

Androgenetic alopecia is a problem not only for adults but also for patients under the age of 16.[5] Diffuse thinning in a young girl or thinning in a female pattern in a male adolescent can make the diagnosis of AGA challenging. Nowadays, the number of young patients with AGA is growing, and interest in the disease is increasing.

Androgenetic alopecia is a progressive disease. The prompt diagnosis and early start of the treatment noticeably enhance the outcome. Trichoscopy is an important tool for formulating a diagnosis of hair and scalp disorders.

With the help of trichoscopic examination, we are able to evaluate the perifollicular and interfollicular skin surfaces, blood vessels, and hair shafts (Table 1). Trichoscopy has many profits. It is not painful, is non-invasive, and is a rapid method that gives the opportunity to assess the whole research area. In case of an unspecified diagnosis, using trichoscopy, it is possible to determine the area where the biopsy and histological research should be done.[6,42]

For a trichoscopic examination, it is important to see the whole scalp and all locations of the body covered with hair, in spite of being sure about the diagnosis. For example, in patients with AGA, even when we clearly see the specific trichoscopic features of the disease, the coexistence of scarring alopecia is still possible because this type of alopecia at an early stage may be invisible to patients and may not cause significant discomfort. Also, if we see any typical signs of cicatricial alopecia, we have to check hairy areas, for example, armpits and groins, to exclude body involvement.

Table 1 Trichoscopic features typical for the diseases clinically mimicking androgenetic alopecia

Diseases	Trichoscopic features absent in androgenetic alopecia
Telogen effluvium	• Upright regrowing hairs • Lack of the difference of hair shaft thickness and number of hairs in follicular units between frontal and occipital area
Lichen planopilaris	• Perifollicular erythema and scaling • Scales entangling hair shafts up to 2–3 mm above the scalp surface in a tubular manner • Hair casts • Violaceus areas • Dystrophic hairs • Absence of follicular openings • Fibrotic white dots • White areas • Milky red areas • Absence of vellus hairs
Fibrosing alopecia in pattern distribution	• Loss of the follicular openings • Perifollicular erythema • Perifollicular scaling • Perifollicular white halo
Central centrifugal cicatricial alopecia	• Peripilar white-gray halo • Irregular distribution of pinpoint white dots • Irregular white patches
Frontal fibrosing alopecia	• Absence of vellus hairs • Minor perifollicular scaling and erythema • Lack of follicular openings • Ivory-colored background • Pili torti on hair-bearing edge • Lonely hairs
Traction alopecia	• Perifollicular erythema and atypical red vessels • Hair casts • Black dots • Broken hairs • Coiled hairs
Trichotillomania	• Hairs broken at different lengths • Coiled hairs • Hook hairs • Trichoptilosis • Flame hairs • Tulip hairs • V-sign
Alopecia areata	• Exclamation mark hairs • Pigtail hairs • Dystrophic hairs • Black dots and broken hairs

Trichodynia, which is the symptom of diffuse or spotty tenderness, tingling, crawling, itching, burning, and uncomfortable awareness of the scalp, may present in AGA.[25,66] Similar symptoms may exist in cicatricial alopecias. On trichoscopy, the absence of typical features of cicatricial alopecias makes it possible to exclude them. Trichoscopically, only the presence of the features of AGA confirms the fact that it is trichodynia.

Evaluating pseudotrichoscopic signs has to be underlined too; it is connected with hair dyeing and using various hair products. For example, pitfalls, when some artifacts simulate hair disorders and cause misdiagnosis. The most important pitfalls are scalp deposits (dirty dots that may look similar to the dots that we see in alopecia areata), scalp staining (hair dye looking similar to hyperpigmentation), and shaft deposits (dry shampoos or hair styling products looking like hair casts). Therefore, it is important to take a thorough history together with trichoscopy, which is the key to diagnosis.[67]

As AGA is a progressive disease, there is a need for constant treatments and check-ups. With trichoscopy, it is also possible to monitor the progression of the process and the treatment outcome.

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