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Fundamentals of Hair & Scalp Dermoscopy



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CHAPTER 4

Nonscarring Alopecias

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4.1 Androgenetic Alopecia or Male-Pattern Hair Loss

Patricia Damasco, Giselle Martins, Isabella Doche

MALE-PATTERN HAIR LOSS

Male-pattern hair loss (MPHL) is a common condition characterized by progressive thinning of scalp hairs. The disease occurs in all races, although the prevalence in Caucasians is little higher comparing to Asians and African descents.

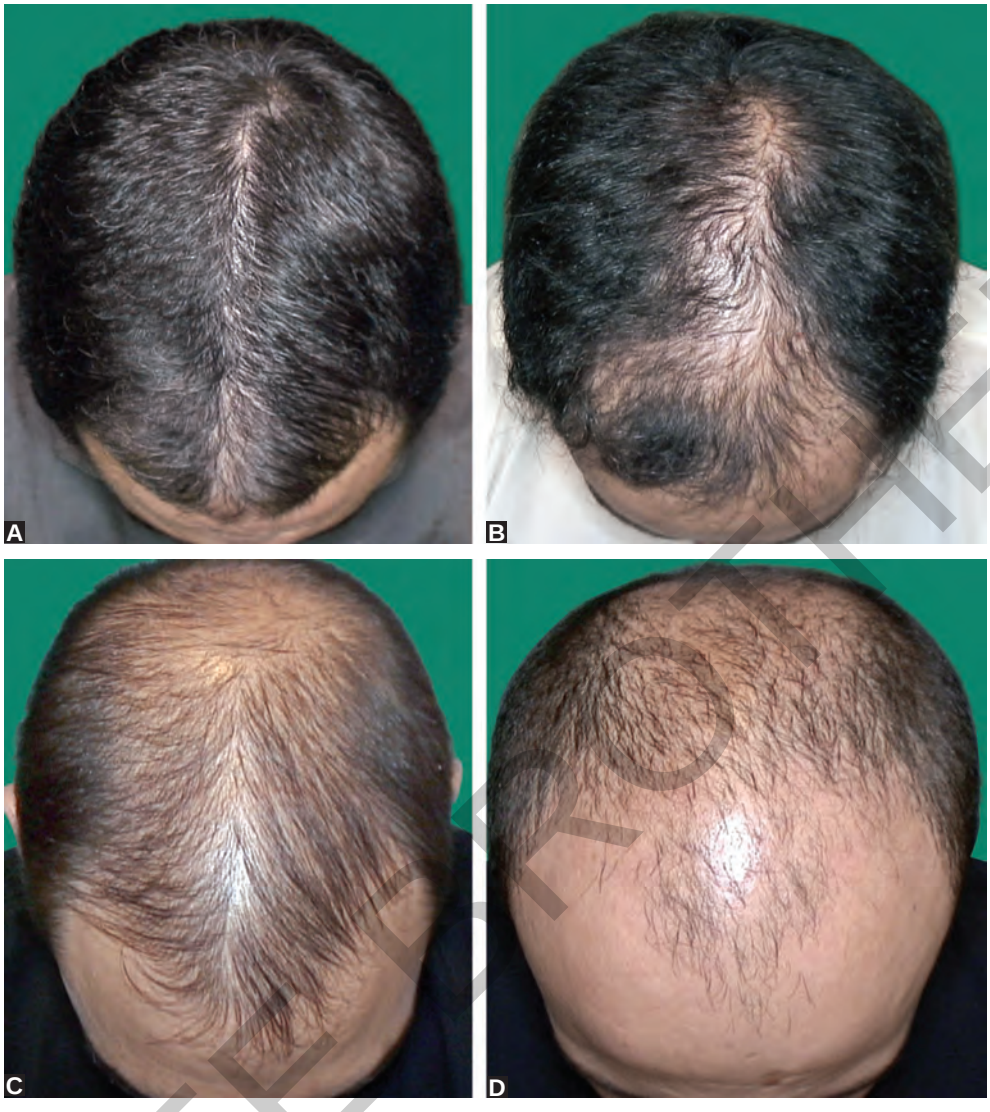
Pathogenesis if MPHL is related to increased conversion of testosterone to dihydrotestosterone by the 5-α-reductase type II enzyme, leading to a progressive hair miniaturization in androgen-sensitive hair follicles. Also, there is a continuous reduction in the duration of the anagen phase over the course of successive hair cycles. The length of the telogen phase remains constant or is prolonged and, over time, the anagen phase becomes so short that the hairs fail to achieve sufficient length to reach the surface of the skin, resulting in empty follicular openings.

Most affected men present with bilateral frontotemporal recession and decreased hair density in the frontal and vertex scalp areas (Hamilton–Norwood Classification) (Figs. 4.1.1A to D).

Dermoscopy shows increased hair shaft diameter diversity (more than 20%), variable yellow dots, peripilar sign (perifollicular brown discoloration), increased proportion of single follicular units and vellus hairs (Figs. 4.1.2 to 4.1.6). In severe cases, scalp pigmentation with a honeycomb-pattern appearance and pinpoint white dots are usually seen (Fig. 4.1.7).

Topical minoxidil and oral finasteride (5-α-reductase type II inhibitor) are the only approved treatments. Hair transplantation can be an option for severe cases.

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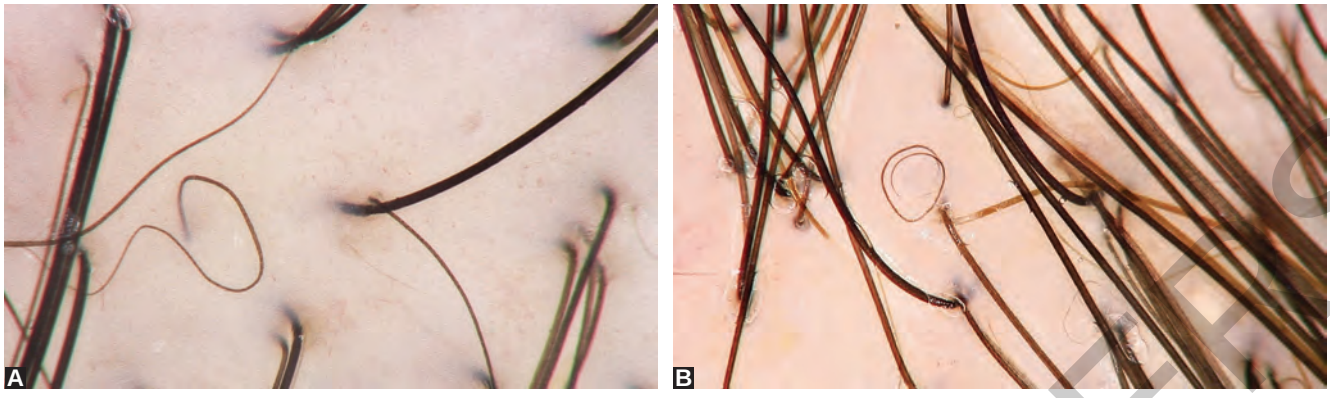


Figs. 4.1.1A to D: Male-pattern hair loss. (A) Frontotemporal recession with mild thinning in the frontal and mid-scalp areas (Norwood–Hamilton II). (B) Moderate frontotemporal recession (Norwood–Hamilton type III). (C) Diffuse hair thinning, mostly on the vertex scalp area (Norwood–Hamilton V). (D) Severe diffuse hair thinning (Norwood–Hamilton VI).



Fig. 4.1.2: Early male-pattern hair loss. Mild decreased hair density and increased hair diameter variability in the vertex scalp from an African-descent patient. Some areas present a more prominent honeycomb pattern.

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Figs. 4.1.3A and B: Early male-pattern hair loss. Circle or pigtail hairs and wavy hairs are regrowing hairs that appear as thin, isolated coiled hairs surrounded by terminal and intermediate hairs.

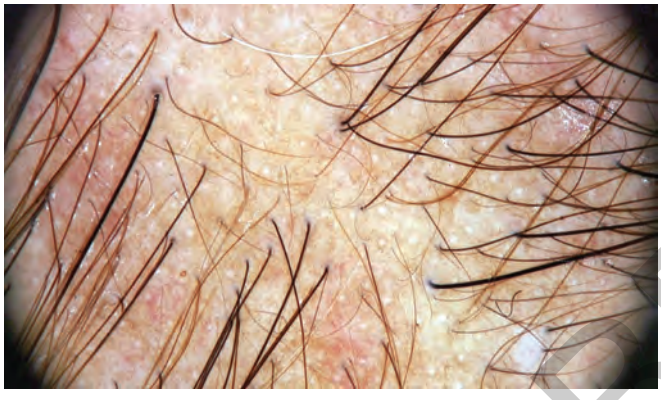


Fig. 4.1.4: Moderate male-pattern hair loss. Increased hair shaft thickness variability. Note thin, intermediate, and thick hairs simultaneously in the field.

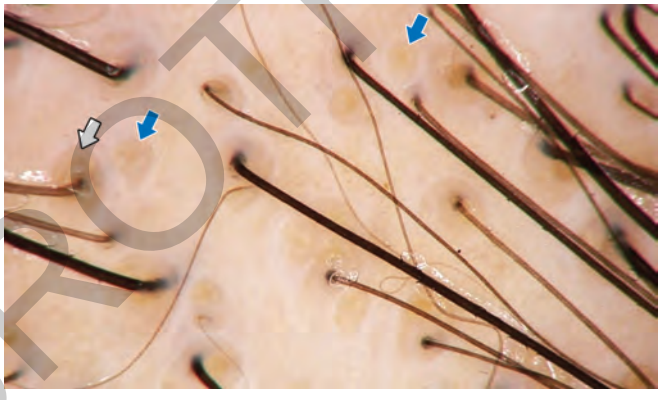


Fig. 4.1.5: Moderate male-pattern hair loss. Multiple small yellow dots (blue arrows) filled with keratotic material and devoid of hair shafts. Note large yellowish oily hues (white arrow) caused by the remains of sebaceous debris encircling terminal hairs.

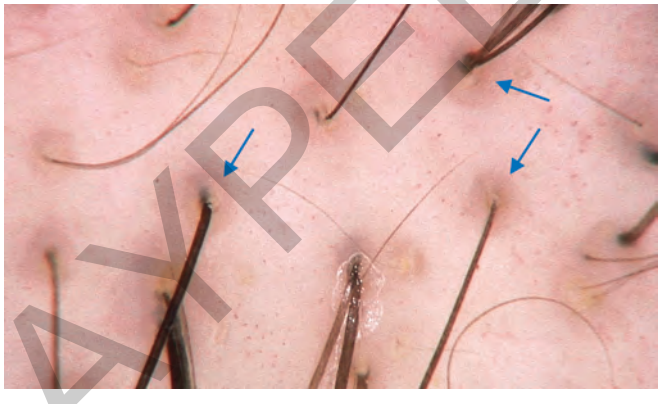


Fig. 4.1.6: Moderate male-pattern hair loss. Vellus hairs and peripilar sign (blue arrows), a slightly depressed brownish halo surrounding some terminal hairs.

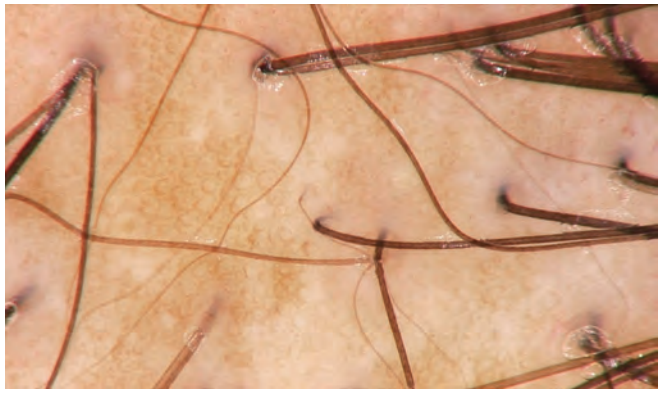


Fig. 4.1.7: Severe male-pattern hair loss. Honeycomb pattern due to chronic sun exposure and pinpoint white dots.

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Take-Home Message

- Male androgenetic alopecia is a very frequent condition with progressive thinning of frontal and mid scalp hairs.
- Follicular miniaturization is the hallmark of the disease.
- Dermoscopy shows increased hair diameter variability, variable yellow dots, peripilar sign, increased vellus hairs, and single follicular units.
- Honeycomb network can be seen in more severe cases.

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4.2 Female-Pattern Hair Loss

Giselle Martins, Patricia Damasco, Isabella Doche

FEMALE-PATTERN HAIR LOSS

Female-pattern hair loss (FPHL) is a diffuse nonscarring alopecia affecting women with scalp hair thinning and miniaturization. In fact, around 40% of women by the age of 50 years are affected by FPHL. The exact etiology of FPHL still remains uncertain, although a polygenic inheritance mode with genes inherited from both parents is acceptable. As it is not clear if androgens play a putative role in this kind of hair loss, the term FPHL is preferred instead of "female androgenetic alopecia."

Female-pattern hair loss presents quite differently from male-pattern hair loss (MPHL), which usually begins with a receding frontal hairline that progresses to a bald area over the vertex scalp (Figs. 4.2.1A to C). These signs are very uncommon in woman, unless hyperandrogenism is present. Usually, patients with FPHL present with diffuse hair thinning and decreased hair density (Ludwig Classification). Some patients complain of increased hair shedding, which should be differentiated from telogen effluvium and alopecia areata incognita.

Dermoscopy of the anterior scalp shows the same features as in MPHL. An increased proportion of vellus and fine hairs (hair diameter variability), yellow dots, perifollicular discoloration (peripilar sign), isolated circle and wavy hairs, and follicular units with only one hair shaft are commonly seen (Figs. 4.2.2 and 4.2.3). In long-standing cases, honeycomb pattern, pinpoint white dots, and focal atrichia can be seen (Fig. 4.2.4). It is very important to compare the anterior and posterior scalp hairs, as temporal hairs are normally thinner and shorter.

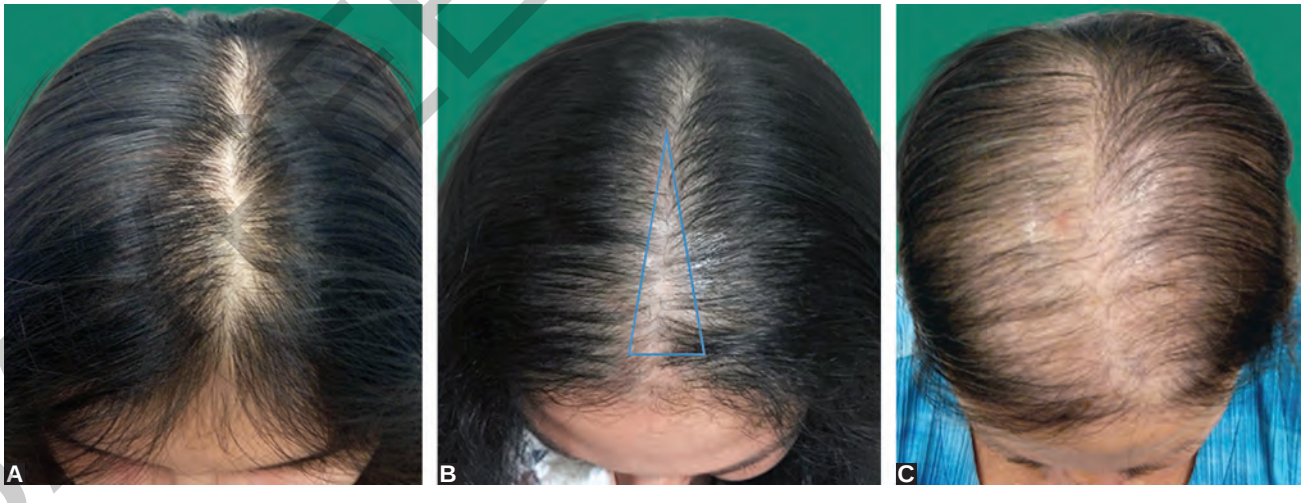
Recently some trichoscopic criteria were described by Rakowska et al. and have been used to help the early diagnosis of FPHL with 98% of specificity.

MAJOR CRITERIA

1. More than four yellow dots in four images at 70X in the frontal area.
2. Lower average hair thickness in the frontal area in comparison with the occiput (calculated from not less than 50 hairs from each area).
3. More the 10% of thin hairs (below 0.03 mm) in the frontal area.

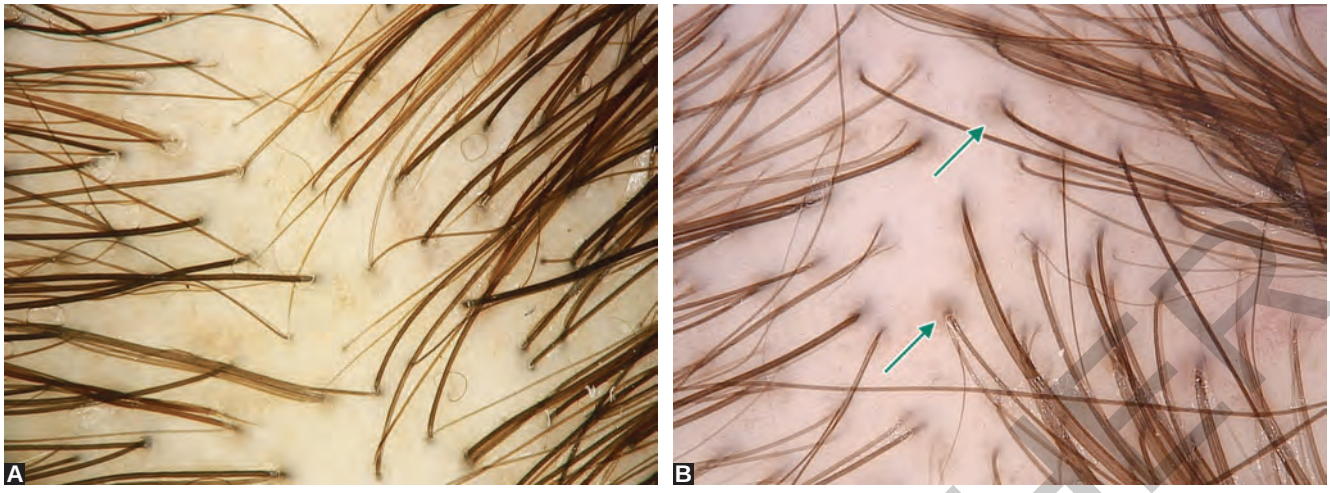
MINOR CRITERIA

1. A ratio of single-hair unit percentage in the frontal area to the occiput greater than 2:1.
2. A ratio of number of vellus hairs in the frontal area to the occiput greater than 1.5:1.

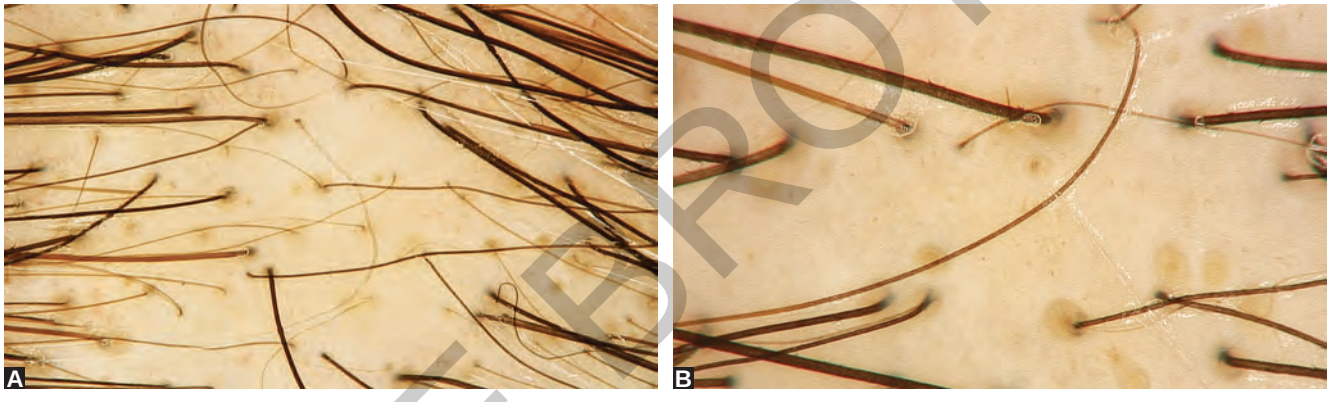


Figs. 4.2.1A to C: Female-pattern hair loss. (A) Mild frontal hair thinning (Ludwig I). (B) Moderate hair thinning, forming the "Christmas-tree pattern" (blue triangle) (Ludwig II). (C) Severe and diffuse hair thinning (Ludwig III). Note focal atrichia with small alopecic areas. After making a central parting, note the maintenance of the frontal hairline despite the severity of the disease.

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Figs. 4.2.2A and B: Mild female-pattern hair loss. (A).Hair shaft variability (>20%), isolated circle hairs, and single follicular units (hair follicles with one hair shaft) in the frontal scalp area. (B) Peripilar sign (green arrows), a slightly depressed brownish follicular halo can sometimes be seen surrounding some terminal and intermediate hair follicles in early cases. It corresponds to the mild infundibular inflammation and tends to disappear after treatment.



Figs. 4.2.3A and B: Moderate female-pattern hair loss. (A) Decreased hair density, increased hair diameter variability with multiple vellus and fine hairs. (B) Yellow dots irregularly distributed in the scalp and increased hair diameter variability.



Fig. 4.2.4: Severe female-pattern hair loss. Focal atrichia that corresponds to small alopecic areas with preserved follicular openings, vellus, and short hairs.

3. A ratio of hair follicles with perifollicular discoloration in the frontal area to occiput greater than 3:1.

Fulfillment of two major criteria or one major and two minor criteria is required to diagnose FPHL based on trichoscopy.

Female-pattern hair loss (FPHL) should be differentiated from senescent or senile alopecia, which normally occurs in patients over 60 years of age. Irregular areas of central hair loss, perifollicular scale, and perifollicular erythema may suggest fibrosing alopecia in a pattern distribution, a scarring process.

As MPHL, FPHL has no cure and it is very important to manage expectations when starting treatment, as the main aim is to stop or slow hair thinning, and not promoting hair regrowth. Anti-androgens drugs (e.g. spironolactone, cyproterone, finasteride) associated with topical minoxidil (2% solution or 5% foam) can be used with variable results.

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Take-Home Message

- Dermoscopy of frontal and vertex scalp areas shows increased hair shaft diameter variability (>20%), yellow dots, vellus hairs, short regrowing hairs, isolated and few circle or wavy hairs.
- Peripilar sign, single follicular units, and vellus hairs in the frontal scalp indicate early androgenetic alopecia.
- Long-standing disease shows honeycomb pattern, pinpoint white dots, and areas with focal atrichia.
- Main differential diagnosis includes senescent or senile alopecia, alopecia areata incognita, telogen effluvium, and fibrosing alopecia in a pattern distribution.

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4.3 Senescent or Senile Alopecia

Isabella Doche

SENILE ALOPECIA

Senescent or senile alopecia (SA) is the diffuse and permanent scalp hair thinning in patients over 60 years with no family history of androgenetic alopecia (AGA). Patients present with diffuse scalp thinning and decreased hair volume (Fig. 4.3.1). Dermoscopy shows diffuse decreased hair thinning and density, and multiple hair shafts with one follicle, mostly on the top of the scalp. Hair diameter variability and yellow dots are usually absent (Fig. 4.3.2).

Although SA and AGA may coexist in the same patient, the concept of whether SA and AGA are distinct disorders remains controversial.

Microarray comparison of age-matched subjects with AGA, SA, and normal controls without hair loss has shown differences in gene expression profiles, suggesting that AGA and SA may represent two independent hair disorders and that nonandrogen pathways may also contribute to hair loss. AGA genes related to follicle homeostasis and hair cycling were underexpressed compared to SA genes. Also, the androgen receptor is upregulated in AGA, but not in SA. In contrast, SA revealed changes in genes important to apoptosis processes.

These genetic differences can have significant implications in terms of treatment of hair loss at different ages. Topical treatments, such as minoxidil and laser devices can benefit patients with senescent or age-related thinning, although beneficial results with finasteride remain ascertained.



Fig. 4.3.1: Senescent alopecia. Diffuse hair thinning in a 68-year-old female patient.



Fig. 4.3.2: Senescent alopecia. Decreased hair density and multiple single hair units (follicular units with only one hair shaft) on the vertex area of the scalp. Note the lack of hair diameter variability, no thin or vellus hairs, and no yellow dots.

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Take-Home Message

- Senile alopecia is the diffuse hair thinning in people older than 60 years and no family history of androgenetic alopecia.
- AGA and SA can coexist in the same patient.
- Although SA and AGA show different gene profile expression, there is no consensus on whether SA is in the spectrum of AGA.
- Dermoscopy shows diffuse decreased hair thinning and density that can be more pronounced on the vertex area.
- Absent or mild hair miniaturization and variability with a few vellus hairs can be often present.

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4.4 Telogen Effluvium

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TELOGEN EFFLUVIUM

Telogen effluvium (TE) is a nonscarring and diffuse hair loss. Acute TE occurs usually 3 months after a triggering event and is self-limiting, lasting about 6 months. Many potential triggers have been implicated in the pathogenesis of TE, including starting and stopping sex hormone therapy, parturition, major illness, surgery, rapid weight loss or poor nutrition, thyroid dysfunction, high fever, hemorrhage, and acute psychosocial stressors. In acute cases, there is severe hair shedding with variable decrease in hair density. Hair pull test is usually positive with more than 6 telogen hairs.

Chronic telogen effluvium (CTE) is an idiopathic condition, presenting with diffuse scalp hair loss and a fluctuating course that persists for more than 6 months, without any widening of the central part of the scalp or follicular miniaturization (Figs. 4.4.1 and 4.4.4). Although some cases of CTE may follow TE with a known trigger, in most cases, a specific trigger cannot be identified. It typically affects middle-aged women presenting with insidious onset and alternating periods of spontaneous remissions and relapses that can last for several years. Chronic hair loss may produce diffuse thinning of scalp hairs, decreased hair volume, and bitemporal recession (Fig. 4.4.1B). Some patients may refer a painful or burning sensation in the scalp (trichodynia). Shorter and regrowing frontal hairs may be seen in patients with resolving effluvium (Fig. 4.4.1C). Hair pull test is usually negative.

Dermoscopy is usually inespecific. However, some short and upright regrowing hairs may be found (Fig. 4.4.2). Hair shaft variability can be seen in patients with associated androgenetic alopecia (Fig. 4.4.3). Club hairs, a hair in the final stage of a telogen phase, can be rarely found (Fig. 4.4.5). Trigger causes should be identified and treated specifically. However, in many cases a causal factor cannot be identified.



Figs. 4.4.1A to C: Chronic telogen effluvium. (A) No evident hair thinning. (B) Note the temporal recession. (C) Hair regrowth after treatment.

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Fig. 4.4.2: Acute telogen effluvium. Note a short or upright regrowing hair of normal thickness.



Fig. 4.4.3: Acute telogen effluvium. Multiple short and upright regrowing hairs of normal thickness, vellus hairs, yellow dots, and hair shaft variability in a patient with associated mild androgenetic alopecia.



Fig. 4.4.4: Chronic telogen effluvium. Note the absence of hair shaft variability.

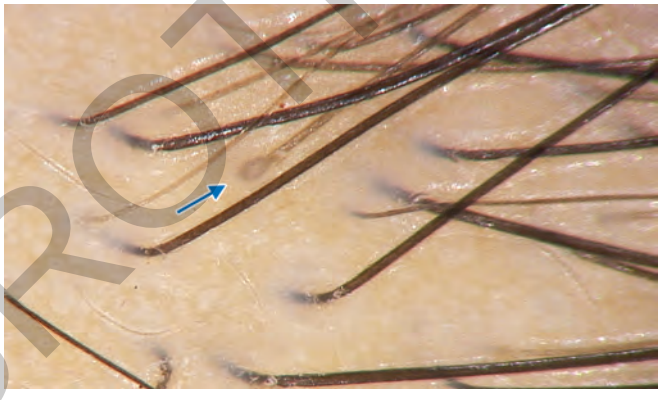


Fig. 4.4.5: Telogen effluvium. Club hairs (blue arrow) can be rarely found in telogen effluvium. They are the final product of a hair in the telogen stage.

Take-Home Message

- Telogen effluvium is a diffuse and nonscarring hair loss.
- Acute telogen effluvium usually occurs 3 months after a triggering event, lasting up to 6 months. Severe shedding and variable decrease in hair density are present.
- In chronic telogen effluvium, diffuse shedding has a fluctuating course and lasts more than 6 months. Bitemporal recession, decreased hair volume and variable hair shedding can be noted.
- Trichoscopy is not specific. Short regrowing hairs may be seen in acute and chronic cases.

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4.5 Alopecia Areata

Isabella Doche, Maria K Hordinsky

ALOPECIA AREATA

Alopecia areata (AA) is a chronic, immune-mediated nonscarring alopecia that attacks anagen hair follicles. The disease affects around 2% of the population, both male and female patients at all ages. Clinically, it may appear as round or oval unique or multiple patches, diffuse scalp hair loss (alopecia totalis), diffuse scalp and body hair loss (alopecia universalis), or ophiasis pattern (alopecia in the lower occipital scalp and the region above the ears) (Figs. 4.5.1A to D). Patchy lesions may mimic tinea capitis, traction alopecia, loose anagen syndrome, aplasia cutis congenita, and pseudopelade (Fig. 4.5.1E). Other causes of acute alopecia may be ruled out in diffuse and extensive cases. Hair-regrowing lesions may show hypopigmented hair shafts (Fig. 4.5.1F).

When the AA is active, pull test is usually positive with dystrophic hairs. Partial or total eyebrow alopecia and nail abnormalities may occur, and nail pittings are the most reported feature.

Dermoscopy of active lesions shows exclamation mark hairs, broken hairs, vellus hairs, yellow dots, and black dots (Figs. 4.5.2 to 4.5.4) Caudability hairs, which is a long exclamation mark hair, are usually seen in the border of enlarging patches (Fig. 4.5.4B). Pseudomonilethrix/monilethrix-like hairs with irregular Pohl–Pinkus



Figs. 4.5.1A to F: Alopecia areata. (A) Unique alopecic patch. (B and C) Ophiasis pattern. (D) Alopecia totalis with areas of hair regrowth. (E) Long-standing patch mimicking a scarring process. (F) Diffuse alopecia presenting hair regrowth and hypopigmented hair shafts.

Source: (A, B, E, F) Division of Dermatology, Hospital das Clínicas, University of São Paulo Medical School. (D) Dr Giselle Martins.

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Fundamentals of Hair & Scalp Dermoscopy

The aim of this book is to provide the most updated information on hair and scalp dermoscopy, also known as trichoscopy. This method has been increasing in popularity, as a very practical, quick and useful tool in the study of hair disorders. The text assesses detailed information in a concise, practical and easy-to-ready manner for expert dermatologists and young practitioners. The first part comprises basic knowledge on normal and abnormal trichoscopic structures and on the most popular devices available in the market. The second part discusses detailed clinical and trichoscopic description of each disease using evidence-based information. In the end of each chapter, a "take home message box" is included to help memorizing the most important features regarding the disease and the corresponding topic. The last chapter comprises a special session on the most frequent pearls and pitfalls in trichoscopy according to the clinical experience of the authors and some tips to enhance the examination in the daily practice. Many world recognized experts in this field were invited to contribute in this project.

Isabella Doche MD PhD is a dermatologist expert in trichoscopy from Sao Paulo (Brazil). She started her PhD thesis on hair diseases at the University of Minnesota (USA) and completed at University of Sao Paulo (Brazil). Since 2009, she has been working in joint hair researches in both universities. She is member of the Brazilian Society of Dermatology, American Hair Research Society and European Hair Research Society and reviewer of the Journal of American Academy of Dermatology. She helps to organize conferences and coordinate the Department of Hair and Nails of the Brazilian Society of Dermatology in Sao Paulo (Brazil) and the American Hair Research Society. Nowadays, she practices trichoscopy in the Hair Diseases Clinic of the University of Sao Paulo (Brazil) and at her private practice. She has been invited to speak in the most important national and international meetings on hair diseases and to chair sessions in the European Hair Research Society and in the World Congress for Hair Research. She is the author and co-author of publications about hair and scalp diseases.

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