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The Alopecias

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2nd Edition

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CHAPTER 5

Physiological Alopecia

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CONDITIONS

Physiological alopecia includes three conditions, namely:

1. Physiological alopecia in an infant
2. Hair loss in adolescent boys
3. Androgenetic alopecia (AGA)

PHYSIOLOGICAL ALOPECIA IN AN INFANT

INTRODUCTION

Physiological alopecia was first described in 1907 by Brocq. It is commonly seen in 2–3 months old babies but can be seen right from the first day of birth. Studies have shown a prevalence of 9–12%. It is more common in Caucasian infants. Gender incidence is equal.

ETIOLOGY

Delayed anagen release: The fetal scalp hair development starts at the 3rd–4th month of gestation and anagen hair covers the scalp by the 18th–20th week of gestation and at 26–28 weeks of gestation, the hair passes through catagen and telogen from the frontal to the parietal areas; however, the occipital hair remains in anagen phase and does not go through this process due to a signal in normal full-term pregnancies. The telogen is delayed till term and subsequently, the hair enters telogen suddenly and hair is shed 2–3 months after birth.

Localized pressure has also been suspected to be an etiologic factor but has subsequently been ruled out.

Studies have shown that it occurs in a normal pregnancy state, such as mother's age < 34 years and in normal full-term deliveries. This further supports the theory that it is a physiological phenomenon.

CLINICAL FEATURES (FIGS. 1 TO 15)

It usually starts as an oval- or band-shaped patch of alopecia over the occipital region and subsequently may progress to involve the whole scalp over a period of weeks. It usually starts in the 2nd–3rd month of life.

The lower margin of the patch is well defined and the upper border merges with hair over the vertex. A band of hair may present below the patch extending to the nape of the neck. The alopecia is reversible and regeneration usually occurs within the first year of life.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis includes pressure alopecia, alopecia areata, and halo scalp ring.



Fig. 1: Onset of physiological alopecia



Fig. 4: The lateral view of figure 3



Fig. 2: Progress of alopecia



Fig. 5: Note the hair shafts of different lengths



Fig. 3: Established case of physiological alopecia



Fig. 6: Regrowth of physiological alopecia

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Fig. 7: Note multiple irregular patches of physiological alopecia



Fig. 10: Patterned form of physiological alopecia



Fig. 8: Case of physiological alopecia, which was mistaken for pressure alopecia



Fig. 11: Different view of figure 10 mimicking temporal triangular alopecia



Fig. 9: Closer view of figure 8



Fig. 12: Left lateral view of figure 10

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Fig. 13: Physiological alopecia over the occipital area mimicking pressure alopecia



Fig. 15: Lateral view of patient in figure 14



Fig. 14: Physiological alopecia having a tumid appearance



Fig. 16: M-shaped hairline seen in adolescent boys along with acne

MANAGEMENT

The hair loss is reversible and grows back within 1 year. The parents and guardians need to be reassured.

HAIR LOSS IN ADOLESCENT BOYS

Children have a straight hairline. At puberty, the male sex hormones cause recession over the frontal area of the scalp. The hair loss occurs as two triangular areas on both sides of the midline, leading to an M-shaped hairline **(Fig. 16)** and loss of the straight hairline seen in children **(Fig. 17)**. Castrated males retain the straight hairline seen in children.



Fig. 17: A 7-year-old child showing a straight hairline

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ANDROGENETIC ALOPECIA

INTRODUCTION

Androgenetic alopecia is a patterned progressive hair loss, which occurs in genetically predisposed individuals when exposed to androgens. It is due to conversion of susceptible terminal to vellus hairs on androgen mediation. Balding is absent in castrated males, pseudohermaphrodites, and people with androgen insensitivity syndrome, confirming that androgens are necessary for AGA.

It is also known as common baldness, patterned baldness, premature baldness, and male-pattern hair loss or female-pattern hair loss. It is very common and in genetically predisposed individuals, it is considered to be a form of physiological hair loss.

Hair thinning can begin between the second and fourth decade of life and about 50% of the population has thinning before the fifth decade of life. The prevalence is almost 100% in Caucasians and it almost spares the American Indians. Koreans have a lower age of onset of male-pattern baldness and have increased proportion of Ludwig-pattern hair loss.

The pattern of hair loss varies in males and females and hence, the terms male- and female-pattern hair loss are used. A polygenic mode of inheritance has been suggested. Men need few predisposing genes for expression than females because of high physiological levels of androgens. Therefore, a family history of AGA in the mother is a bad prognostic factor. Males have more first-degree relatives with AGA. Family history is present in 20% of males with AGA.

ETIOLOGY AND PATHOGENESIS

Genetic

- AGA has been determined to have a polygenic inheritance because of a wide range of phenotypes and due to high prevalence.
- Deoxyribonucleic acid (DNA) sequence variations include deletion mutations, single nucleotide polymorphisms, insertion mutations, microsatellite repeats, and copy number variations.
- Epigenetic modifications such as hypomethylation and hypermethylation, which are switch off and on gene expression of DNA in gene promoter areas and X chromosome, inactivation may also be seen.
- There are two major risk loci, which are PAX1/FOXA2 locus situated on chromosome 20 and AR/EDA2R locus situated on the X chromosome. HADC9 locus situated on chromosome 7 may also be a susceptible locus.
- Androgen receptor gene STU1 restriction fragment length polymorphism has been noted in >90% of bald men.
- Other genes, which could possibly be involved, include aromatase, 5-reductase, insulin-like growth factor-2, and estrogen receptor alpha genes.

Hormonal

The basis of AGA is conversion of terminal hair follicle to vellus hair follicle. This occurs by androgenic stimulation of the susceptible hair follicles.

Genetic predisposition is more important than the levels of serum testosterone,

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dehydroepiandrosterone (DHEA), free testosterone, dihydrotestosterone (DHT)—testosterone ratio and insulin-like growth factor 1. Even though all of these have been associated with AGA, studies have shown no relation between the levels of hormones and AGA.

Women with hormone-secreting tumors tend to develop AGA though hirsutism is a much more common feature on androgen excess; however, in studies on women with hair loss, there is a variable portion who do not show any clinical or biochemical signs of androgen excess. AGA in women may be due to genetic predisposition or due to nonandrogenetic mechanism **(Flowchart 1)**.

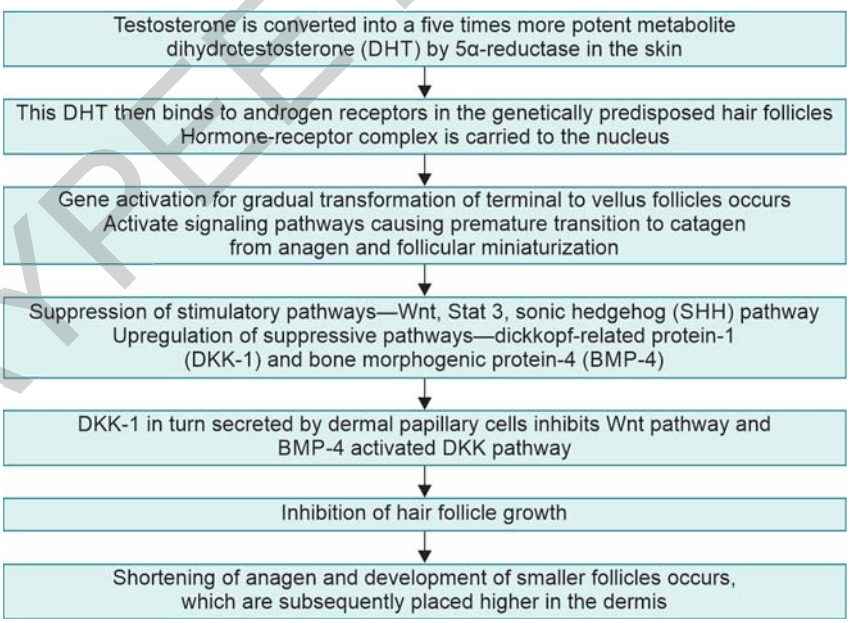
Apart from 5α-reductase, the other enzymes, which can form potent androgens from weak androgens and increase in AGA, include 17β-hydroxysteroid dehydrogenase and 3β-hydroxysteroid dehydrogenase.

The exact roles and processes related to hair follicle stem cells in AGA are not clear. It is considered that while KRT15(hi) stem cells are maintained in bald scalp, there is a defect in conversion of hair follicle stem cells to CD200-rich and CD34+ progenitor cells, both of which are needed to maintain proper follicular activity.

Cytochrome P450 aromatase: It has a protective role on the hair follicles, as it increases conversion of testosterone to estrone and estradiol and; thus, there is less formation of DHT.

Causes for Different Pattern of Androgenetic Alopecia in Women

- Frontal follicles in women contain six times more cytochrome P450 aromatase than men
- Frontal follicles in women have two times the amount of aromatase than occipital follicles
- Women have levels of androgen and 5-reductase



Flowchart 1: Development of androgenetic alopecia

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- There is a difference in distribution of androgen-sensitive follicles
- Estrogen has a protective role in premenopausal women by increasing sex hormone-binding globulin (SHBG) levels and thus reducing the free androgen levels.

Difference in Response of Different Body Areas to Androgens

All hairs are not equally affected by androgens. After puberty, scalp hair is lost; there is growth of body hair and eyebrow hair is not affected. Even on the scalp, the frontal, temporal, and vertex hair are lost, whereas the occipital hair remains intact. In men, the circulating androgens are more than the requirement of hair follicles; therefore, the local factors determine the predisposition and the severity of AGA. In females, since the androgen levels are not high, both the genetic predisposition and androgen levels determine the development and intensity of hair loss.

The hair shows donor dominance; therefore, hair transplanted from the occipital area to the vertex is resistant to AGA and vertex hair when transplanted to the forearm miniaturizes.

The dermal papilla is a group of specialized fibroblasts, which regulates the activity and growth of cells in the hair follicle. Signals from the dermal papillae reach the multipotent epidermal stem cells situated in the hair bulge region causing hair regeneration.

Under the influence of androgens, there is a signal between the dermal papillae and the hair follicle cells, which causes secretion of factors from the dermal papillae. These factors have paracrine action on the hair follicle epithelial cells and autocrine action on the dermal papillae. These factors include cytokines such as interleukin-1 alpha, tumor necrosis factor-alpha, and transforming growth factor beta-1 (TGF-β1) and anagen-maintaining factors such as vascular endothelial growth factor (VEGF), insulin-like growth factor, and basic fibroblast factor.

Decreased expression of anagen-maintaining factors and increased expression of cytokines promotes apoptosis of hair follicles leading to catagen.

Dihydrotestosterone causes upregulation of *DKK-1* gene leading to inhibition of outer root sheath cells, thus leading to apoptosis.

Wnt/β-catenin signaling pathway plays a critical role in maintenance of dermal papillary cell induction for hair shaft growth and hair follicle regeneration. Androgens inhibit this pathway by increasing the glycogen synthase kinase expression.

For proper follicular activity, hair follicle stem cells need to be converted to CD34+ and CD200-rich progenitor.

In AGA, there is a defect in this conversion.

Hair cycle

The anagen duration in AGA decreases with each cycle and the telogen duration remains same or is increased. Thus, the anagen–telogen ratio gets reduced and there is excessive hair shedding. Since the latent or kenogen phase is increased, the number of empty hair follicles increases on the scalp, thus leading to thinning of hair.

Since the duration of anagen determines the length of hair, the length of the hair reduces with each cycle and finally, it is insufficient to reach the skin surface, leading to empty follicular pores.

Hair follicle miniaturization

The density of hair follicles does not change with balding, but there is decrease in the size of follicles, as terminal follicles are gradually converted to vellus hair follicles.

Androgen targets the dermal papilla, which is needed for maintenance of hair growth. The dermal papilla size also determines the hair

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matrix size and thus determines the size of the hair shaft. The size of anagen follicles remains constant throughout one cycle; therefore, the miniaturization occurs in between cycles. Miniaturization of follicles ultimately leaves behind fibrous tracts or streamers or stelae, which are dermal remnants of the follicle. These stelae extend to the miniaturized follicle from the subcutaneous tissue through the follicular tract.

The follicular stelae may show Arao–Perkins bodies. These are seen in the neck of the dermal papilla and are a small cluster of elastic fibers, which clump in catagen and are seen at the lowest point of the follicle. As shortening of the anagen hair occurs, many elastic clumps can be found in the follicular stelae similar to the rungs of a ladder.

CLINICAL FEATURES

The peak age of onset is in the third to fourth decade in males; however, 18% of men have onset at the age of 18–29 years. Females have a bimodal peak age of onset with the first peak in the third decade and the second peak in the fifth decade of life. The onset may be sudden when it begins as telogen effluvium or insidious when hair fails to regrow after falling. The rate of progression and severity of baldness is variable and unpredictable.

Patients with AGA were found to have 3.46 times the risk of developing metabolic syndrome, when compared to controls. AGA patients with metabolic syndrome also have a tendency for worse metabolic profiles. Younger age of onset, female sex, and African ancestry were associated with higher odds of developing metabolic syndrome. Hence, in young patients with severe AGA, appropriate counseling and screening for metabolic syndrome may be warranted.

The factors which determine if a person will become bald are age of onset, rate of progression, pattern of hair loss, and the susceptibility to AGA **(Figs. 18 to 26)**.

Females and males are equally affected, but the hair loss is more prominently seen in males.

The frontal hairline is usually lost in case of males with a few areas of complete baldness, whereas in females, the hairline is preserved with usually only diffuse hair loss without areas of complete hair loss **(Figs. 27 to 30)**. The number of miniaturized hairs is more in males compared to females.



Fig. 18: Classical androgenetic alopecia (AGA) with history of father with AGA



Fig. 19: Classical androgenetic alopecia (AGA) with history of maternal uncle with AGA

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Fig. 20: Left lateral view of figure 19



Fig. 21: Right lateral view of figure 19



Fig. 22: Classical androgenetic alopecia (AGA) with history of maternal grandfather with AGA



Fig. 23: Classical androgenetic alopecia (AGA) without any family history of AGA



Fig. 24: Androgenetic alopecia in a boy < 18 years of age



Fig. 25: Right lateral view of figure 24

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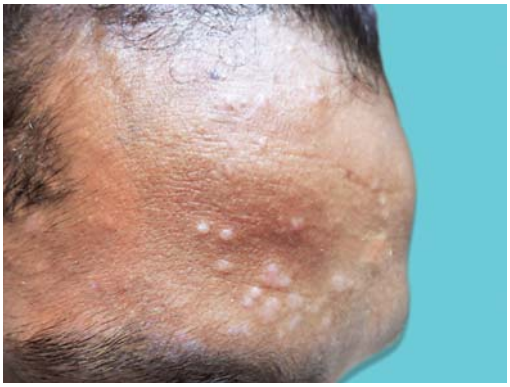

Fig. 26: Case of androgenetic alopecia associated with acne. Note the acne lesions on the forehead



Fig. 29: Perimenopausal woman with androgenetic alopecia



Fig. 27: Early-onset androgenetic alopecia in a girl less than 18 years of age



Fig. 30: Same patient as in figure 29 showing normal occipital hair density



Fig. 28: Androgenetic alopecia in a teenage girl with central partition widening

CLASSIFICATIONS OF ANDROGENETIC ALOPECIA

Female-pattern Hair Loss

Females commonly show Ludwig type of hair loss with diffuse thinning; however, Hamilton type, i.e., frontoparietal type and Olsen's, i.e., Christmas tree pattern of hair loss may also be seen.

Ludwig Scale (Fig. 31)

- Stage 1: Minimal widening of the parting with thinning of hair over the anterior part of the crown

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- Stage 2: More evident thinning of crown with increase in the number of short and thin hairs
- Stage 3: Significant widening of the parting. Crown is almost bald. Frontal hair is maintained.

Sinclair Scale (Figs. 32 to 37)

- Grade 1: Normal—no signs of alopecia
- Grade 2: Widening of the central parting
- Grade 3: Widening of the central parting with thinning hair on both sides of the parting
- Grade 4: Top of the scalp shows diffuse hair loss
- Grade 5: Advanced hair loss

Olsen

It presents as a frontovertical type of hair loss. A triangular type of hair loss is seen with a progressive decrease in density of hair from the vertex moving up to the front of the scalp.

Very rarely, male-pattern type of hair loss may also be seen in females **(Fig. 38)**.



Fig. 31: Ludwig scale stage 2 hair loss progressing to stage 3



Fig. 33: Closer view of figure 32



Fig. 32: Sinclair scale grade 3 hair loss



Fig. 34: Sinclair scale grade 4 hair loss

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Fig. 35: Sinclair scale grade 4 progressing to grade 5



Fig. 37: Adolescent showing recession of hairline along with acne papules



Fig. 36: Top of the scalp shows diffuse hair loss

Ebling and Rook Classification

These are as follows:

- *Type I*: Hair thinning over the crown
- *Type II*: Pronounced thinning of hair over the crown
- *Type III*: Diffuse continued hair loss over the crown and starting of hair loss from the frontotemporal hairline
- *Type IV*: Continued diffuse hair loss and frontotemporal recession
- *Type V*: Complete hair loss over the top of the scalp similar to male-pattern hair loss

Male-pattern Hair Loss

Hamilton Classification (Figs. 39 to 47)

- *Type I*: No recession of hairline
- *Type IA*: The anterior border of hairline is high on the forehead
- *Type II*: Anterior border of hairline has triangular areas or recession in the frontoparietal regions, which are symmetrical and does not extend posteriorly beyond a point which is 3 cm anterior to a coronal line beyond 3 cm in a line drawn between external auditory meatuses in a coronal plane. Hair may be sparse over the midfrontal border, but the hair loss in this area is less than the frontoparietal region
- *Type III*: These include borderline cases.

  It includes scalps in where there are scars, unusual type of sparseness, and denudation lateral asymmetry in which classification is inaccurate.

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Salient Features

- The book "*The Alopecias*" has been diligently crafted for use by the internists and dermatologists.
- It has different sections with several images, which makes diagnosis easy.
- Therapeutic approach has been simplified.
- It carries the most recent developments in alopecia.

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