

Psychological Effect, Pathophysiology, and Management of Androgenetic Alopecia in Men

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Androgenetic alopecia in men, or male pattern baldness, is recognized increasingly as a physically and psychologically harmful medical condition that can be managed effectively by generalist clinicians. This article discusses the clinical manifestations, epidemiology, physical and psychosocial importance, pathophysiology, diagnosis, and management of androgenetic alopecia in men. Androgenetic alopecia affects at least half of white men by the age of 50 years. Although androgenetic alopecia does not appear to cause direct physical harm, hair loss can result in physical harm because hair protects against sunburn, cold, mechanical injury, and ultraviolet light. Hair loss also can psychologically affect the balding individual and can influence others' perceptions of him. A progressive condition, male pattern baldness is known to depend on the presence of the androgen dihydrotestosterone and on a genetic predisposition for this condition, but its pathophysiology has not been elucidated fully. Pharmacotherapy, hair transplantation, and cosmetic aids have been used to manage male pattern baldness. Two US Food and Drug Administration–approved hair-loss pharmacotherapies—the potassium channel opener minoxidil and the dihydrotestosterone synthesis inhibitor finasteride—are safe and effective for controlling male pattern baldness with long-term daily use. Regardless of which treatment modality is chosen for male pattern baldness, defining and addressing the patient's expectations regarding therapy are paramount in determining outcome.

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DHT = dihydrotestosterone; PSA = prostate-specific antigen

Management of androgenetic alopecia in men, a common dermatologic condition also known as male pattern baldness, has historically been outside the scope of the generalist clinician's practice—perhaps primarily because of its perceived inconsequentiality and the lack of nonsurgical strategies for effective management. However, because of ongoing research and recent developments, androgenetic alopecia in men is recognized increasingly as a

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physically and psychologically harmful medical condition in some men^{1,2} that can be managed effectively by generalist clinicians. Therefore, rather than being inconsequential among these men, androgenetic alopecia can be a harmful condition that warrants intervention.

Advances in surgical techniques make hair loss more amenable to treatment than ever before; also, pharmacotherapy is now available that can retard, stop, or partially reverse hair loss, can stimulate some hair regrowth,³ and is safely prescribed on an outpatient basis. With the introduction of effective and tolerable pharmacotherapy, generalist clinicians who are not experts in surgical techniques involving hair transplantation can offer effective intervention.

The general public's increasing knowledge of and readiness to explore pharmacological and surgical solutions to cosmetic problems including baldness has contributed to an upsurge in patient requests to generalist clinicians for intervention options against hair loss.

This article, intended to provide clinicians with the most current information about androgenetic alopecia in men, discusses the clinical manifestations, epidemiology, psychosocial and physical importance, pathophysiology, diagnosis, and management of this condition.

CLINICAL MANIFESTATIONS

Hair loss from androgenetic alopecia in men is progressive and occurs typically in a characteristic pattern, beginning with recession of the frontal hairline and hair loss in the vertex or crown and progressing to complete loss of hair over the frontal and vertex scalp regions.^{4,5} In the most severe form of androgenetic alopecia in men, hair may be present only in a ring around the head in the temporal, parietal, and occipital regions of the scalp. This progression is characterized most often by the 7 categories of the Hamilton-Norwood scale,⁶ which assists in the diagnosis and monitoring of hair loss. Hair loss does not conform to this progression in all individuals.

EPIDEMIOLOGY

The age at onset of androgenetic alopecia in men varies, but occurs on average in men in their mid-20s. The prevalence

and severity of androgenetic alopecia in men increase directly with age. Because male pattern baldness depends on circulating androgens (see “Pathophysiology” section), the condition is not observed in prepubescent children. Androgenetic alopecia is most pervasive among middle-aged to elderly white men.⁷⁻⁹ Approximately 30% of white men are affected by age 30 years, at least 50% are affected by age 50 years, and 80% are affected by age 70 years.⁴ The incidence of androgenetic alopecia also varies with race: white men are more likely to develop baldness than are men of Asian, American Indian, and African heritage. Also, the extent of hair loss often is more extensive in white men than in men of the previously mentioned other ethnicities.⁹

PSYCHOSOCIAL AND PHYSICAL IMPORTANCE

Most men with androgenetic alopecia experience psychosocial effects. Specifically, hair loss affects the balding individual’s feelings of attractiveness and satisfaction with his physical appearance (body image) and can influence other persons’ perceptions of him.^{1,2} The effects of male pattern baldness on self-image and others’ perceptions are not surprising in the context of the importance of hair in the sociocultural context.^{1,2} Hair is an important determinant of physical attractiveness and a means of expressing individuality. Throughout history, abundant hair has symbolized vitality, health, and virility, whereas loss or removal of hair can connote subjugation, loss of individuality, impotency, and/or decrepitude.

The negative effects of hair loss on body image have been observed in several studies of androgenetic alopecia in men.¹⁰⁻¹⁶ Across studies, factors associated with a greater risk of hair loss–related psychological morbidity include young age, not being involved in a romantic relationship, strong reliance on physical appearance as a source of self-esteem, and having preexisting poor self-esteem.² Besides affecting the balding man’s self-image, hair loss can influence others’ perceptions of the balding individual. In studies comparing individuals’ initial impressions to sketches or photographs of balding compared with nonbalding men, balding men were consistently rated as less physically and socially attractive, older, less likable, and less virile.¹⁷⁻¹⁹ However, the degree to which these first impressions of balding men evolve over time has not been studied.

Androgenetic alopecia is not known to be life threatening, but it can lead to physical harm. Hair protects against sunburn, cold, and mechanical injury. Because androgenetic alopecia in men involves loss of the hair’s protection of the scalp from ultraviolet light, it may increase the risk of sunburn and the cellular damage that underlies

skin cancer—possibilities that have not been established empirically.

PATHOPHYSIOLOGY

Normal hair growth occurs at the level of the hair follicle in a 3-phased cycle: (1) anagen, a 2- to 7-year active growth phase during which hair is produced continuously via the division and growth of specialized keratin-producing epidermal cells that surround a dermal papilla at the base of the hair follicle; (2) catagen, a 1- to 2-week transition and involution phase, during which the hair follicle contracts as a result of apoptosis and the hair bulb ascends toward the surface of the skin, loses its root sheaths that anchor the hair in place, and develops a club-shaped end to form a club hair (ie, a hair in the resting state); and (3) telogen, a 5- to 12-week resting phase during which the old club hair is shed. At the end of telogen, germinal cells of the hair follicle once again begin to grow to form a new hair bulb, which becomes the source of a new hair.⁴ On average, in the normal scalp, at least 90% of hairs are in anagen, 1% are in catagen, and 9% are in telogen.²⁰

The basis of androgenetic alopecia in men is a progressive decrease in the density of terminal (thick and pigmented) hairs and a concurrent increase in density of vellus (short, fine, nonpigmented) hairs.²⁰ In effect, terminal hairs are turned off and are transformed into vellus hairs. This effect is attributed to miniaturization of the hair follicle, which is associated with a substantial reduction in hair diameter. Miniaturization may occur abruptly in 1 or a few hair cycles.²¹ In 1 illustrative study of biopsy specimens from 106 men with male pattern baldness and 44 nonbalding control subjects, the ratio of terminal to vellus hairs was 7:1 in the nonbalding scalp compared with 2:1 in the balding scalp.²² In male pattern baldness, the anagen phase shortens, and the telogen phase lengthens or remains the same so that hair length—which depends primarily on the duration of anagen—decreases.²³ Eventually, the hair does not reach the skin surface. Also, the time between the telogen stage and the anagen stage lengthens so that the number of scalp hairs decreases.⁴

Although the mechanisms of these changes have not been established definitively, male pattern baldness is known to depend on androgens—in particular, the androgen dihydrotestosterone (DHT).²³⁻²⁵ Dihydrotestosterone is synthesized from testosterone by 5 α -reductase type 1 and type 2, lipophilic enzymes found on intracellular (nuclear) membranes.²⁴ Type 2 5 α -reductase, expressed in hair follicles and other androgen-dependent tissues such as the prostate gland, appears to be more important than type 1 in male pattern baldness.

Several lines of circumstantial evidence support the crucial role of androgens—and DHT in particular—in male pattern baldness. First, this condition is not observed in eunuchs, who lack androgens; in individuals who lack functional androgen receptors; or in pseudohermaphrodites, who lack 5 α -reductase.^{4,25-27} The absence of baldness in those lacking the gene for 5 α -reductase type 2 suggests a necessary role for DHT. Second, the progression of androgenetic alopecia in men is halted coincident with castration among postpubertal men.⁵ Third, balding scalp contains excessive concentrations of 5 α -reductase, DHT, and the androgen receptor.^{4,28,29} Finally, hair loss is mitigated or inhibited by finasteride, a medication that prevents the conversion of testosterone to DHT by selectively inhibiting the activity of 5 α -reductase type 2.²³ Although the presence of androgens and a genetic predisposition are necessary for androgenetic alopecia in men, much about the pathophysiology of this condition remains to be elucidated.

Androgenetic alopecia in men appears to be inherited, but the mode of inheritance is not yet understood. Hypothesized modes of inheritance include a single autosomal dominant gene, a single pair of sex-linked factors, a dominant gene with increased or variable penetrance in men, and polygenic inheritance.^{5,20} A family history of androgenetic alopecia may be present on either side of the family; however, the absence of such a family history does not exclude the diagnosis.

DIAGNOSIS

Male pattern baldness is diagnosed primarily on the basis of history and physical examination.⁵ Men with a history of progressive hair loss that follows the pattern defined by the Hamilton-Norwood scale are highly likely to have male pattern baldness.

Biopsies can be used as diagnostic aids but seldom are required for diagnosis. Histopathologic changes characteristic of male pattern baldness include a progressive increase in the density of vellus hairs (vellus hair shafts are ≤ 0.03 mm in diameter and thinner than the follicle's inner root sheath), a decrease in the density of terminal hairs (terminal hair shafts are >0.03 mm in diameter and thicker than the follicle's inner root sheath), and a decrease in the ratio of terminal to vellus hair from 7:1 to approximately 2:1.^{5,9} These changes may be observed in the absence of an abnormal total number of hairs per unit area. Androgenetic alopecia is not considered to be an inflammatory condition; however, superficial perifollicular infiltrate may be present.⁵

The differential diagnosis of male pattern baldness includes diffuse alopecia areata—recurrent, nonscarring hair loss that may be associated with autoimmune disease. Un-

like male pattern baldness, alopecia areata typically entails circumscribed and asymmetrical areas of baldness and can involve the eyebrows, face, and other body parts in addition to the scalp. A diagnosis of diffuse alopecia areata is suggested by findings of exclamation-point hairs, pitted nails, and/or a history of periodic regrowth of hair.⁵ Alopecia areata, which is much less common than male pattern baldness, reportedly affects 1.7% of the US population by the age of 50 years.³⁰ Other differential diagnoses include acute and chronic telogen effluvium (ie, excessive shedding of normal club hairs; may be idiopathic or associated with iron deficiency, papulosquamous scalp diseases, or stressors) and early cicatricial alopecia (ie, hair loss arising from the destruction of hair follicles by scarring from processes such as trauma, burns, lupus erythematosus, or lichen planopilaris).

MANAGEMENT

Management of male pattern baldness involves obtaining a medical history, performing a physical examination, assessing changes in scalp hair in the context of the age and occupation of the individual, assessing the importance of hair loss to the patient, and working with the patient to determine the best treatment. Options for managing androgenetic alopecia in men include doing nothing and accepting the cosmetic outcome (the “wait and see” approach), pharmacotherapy, hair transplantation, and cosmetic aids. Hair loss is progressive and does not improve or reverse without treatment.

PHARMACOTHERAPY

Two US Food and Drug Administration–approved pharmacotherapies—minoxidil and finasteride—are available for treatment of male pattern baldness. These medications, which differ in mechanism of action and route of administration, are given as monotherapy or as combination therapy, although few clinical studies of combination therapy have been published to date. These drugs often are prescribed for patients undergoing hair-restoration surgery to reduce the amount of transplanted hair required to meet the patient's objectives and to help the patient maintain a relatively consistent and natural appearance. Although minoxidil and finasteride both retard or stop hair loss and stimulate some hair regrowth, neither medication restores all lost hair or reverses complete baldness. No well-controlled study comparing minoxidil and finasteride has been published to date. In a randomized study in which 99 patients treated with finasteride or minoxidil were monitored for up to 24 months, both agents appeared to be similarly effective for stopping the progression of androgenetic alopecia.³¹

Minoxidil. Initially introduced in the 1970s as a systemic treatment of hypertension, minoxidil now is marketed also as topical 2% and 5% solutions for androgenetic alopecia in men and women.³² Minoxidil is a potassium channel opener, and its mechanism of action in male pattern baldness is unknown. Minoxidil appears to increase the duration of the anagen phase, and its angiogenic effects reverse miniaturization of hair follicles. In placebo-controlled clinical studies, minoxidil slowed hair loss and increased hair density, measured by target-area hair counts, expert panel review of global photographs, and hair weight.^{33,34} Growth of hair appears to peak approximately 4 months after initiation of therapy. The 5% solution is associated with an earlier and more robust response than the 2% solution for male pattern baldness. In a randomized, double-blind, placebo-controlled study in which patients applied 5% minoxidil (n=157), 2% minoxidil (n=158), or placebo (n=78) twice daily, hair density improved more with active treatment than placebo. In addition, androgenetic alopecia improved more with the 5% solution compared with the 2% solution, reflected in target-area hair count increases after 48 weeks of treatment (18.6/cm² for the 5% solution, 12.7/cm² for the 2% solution, and 3.9/cm² for placebo) and in expert panel review of global photographs after 1 year (increased growth in 57.9% of men with the 5% solution, 40.8% of men with the 2% solution, and 23.2% of men with placebo).³³

To maximize efficacy, minoxidil should be applied evenly to the entire affected area of the scalp. Patients should avoid wetting the scalp for at least 1 hour after minoxidil administration to allow the drug sufficient time to be absorbed; also, patients should apply minoxidil before any use of hair gel or hair spray so that absorption is not affected.⁹ Minoxidil must be applied daily to maintain effectiveness. If treatment is discontinued over a period of a few months, the scalp appears to revert to the state that it would have been in without pharmacotherapy.³⁵

Generally, minoxidil is well tolerated with long-term daily use. Adverse events are primarily dermatologic and include irritant contact dermatitis and, less often, allergic contact dermatitis.³⁶ Transient and self-limiting telogen effluvium may begin approximately 3 to 5 weeks after initiation of treatment. Patients should be informed about the possibility of temporary telogen effluvium and advised to continue treatment should it occur.

Finasteride. Initially introduced in a 5-mg dose for treatment of benign prostatic hyperplasia, finasteride is now marketed in a 1-mg dose for treatment of male pattern baldness. Finasteride selectively inhibits the type 2 5 α -reductase isoenzyme responsible for converting testosterone to DHT, the putative hormonal modulator of androgenetic alopecia in men. Finasteride reduces serum and scalp

DHT concentrations by approximately 60% to 70%.³⁷ Finasteride may inhibit or reverse miniaturization of hair follicles as shown by a trend toward improvement in the terminal-to-vellus ratio in a scalp biopsy study.³⁸

The clinical efficacy of finasteride has been documented in well-controlled clinical trials that monitored men with male pattern baldness for up to 5 years. In 2 double-blind placebo-controlled clinical trials that were continued as 4 consecutive 1-year, placebo-controlled extension trials (for a total of up to 5 years of observation), 18- to 41-year-old men with primarily vertex hair loss received daily treatment with 1 mg of finasteride or placebo.³⁹⁻⁴¹ Compared with placebo, finasteride slowed hair loss and increased hair density and length across several efficacy measures. Finasteride was associated with increased target-area hair counts (16.9/cm² vs -4.1/cm² with placebo at 1 year) and higher incidence of increased hair growth (at 1 year, 48% increase with finasteride vs 7% increase with placebo; at 2 years, 66% increase with finasteride vs 7% increase with placebo), effects that were sustained throughout the 5-year treatment period. By photographic assessment, improvement in hair growth or no visible hair loss was observed in 90% of men treated with finasteride (48% improvement; 42% no visible loss) compared with 25% of placebo-treated patients (6% improvement; 19% no visible loss). Growth of hair did not appear to plateau until approximately 1 to 2 years after initiation of therapy. Visible worsening in scalp hair was reported for 10% of finasteride-treated patients compared with 75% of placebo-treated patients after 5 years of treatment. At the end of this 5-year period, hair counts improved in a 1-inch-diameter area of scalp hair loss (from a baseline of 876 hairs) by 277 hairs with finasteride compared with placebo; furthermore, 65% of finasteride-treated patients compared with 0% of placebo-treated patients had increased hair count compared with baseline. Finasteride also was effective vs placebo for slowing hair loss and increasing hair density in a 1-year double-blind study of finasteride in men with predominantly frontal hair loss.⁴²

These objective improvements were associated with increased patient satisfaction and body image.²³ Most finasteride-treated patients reported that they were satisfied with the overall appearance of their scalp at the end of 5 years of treatment. Finasteride-treated men rated their hair growth and satisfaction with their appearance more positively than did placebo-treated men.

Baldness progresses with discontinuation of finasteride as DHT levels return to pretreatment levels. Therefore, to maintain therapeutic benefits, finasteride is taken long-term on a daily basis.

Finasteride predictably reduces total serum prostate-specific antigen (PSA), an effect that appears to plateau

after approximately 6 months of therapy.⁴³ By multiplying the patient's PSA level by 2, the utility of serum PSA as a screening tool for prostate cancer is preserved in men taking daily finasteride.⁴³

Finasteride generally is well tolerated with long-term daily use. Its adverse event profile is comparable to that of placebo except for sexual adverse effects, which were reported slightly more frequently with finasteride use than with placebo over 1 year (decreased libido, 1.8% vs 1.3%; erectile dysfunction, 1.3% vs 0.7%; ejaculatory disorders, 1.2% vs 0.7%).⁴¹ Sexual adverse effects of finasteride often diminish or resolve with continued treatment and reverse on discontinuation of treatment. The incidence of each of the previously cited sexual adverse effects for 1 year of treatment decreased to 0.3% or less with continued treatment with finasteride for up to 5 years.⁴¹

In the Prostate Cancer Prevention Trial, a randomized, double-blind, placebo-controlled trial in which 18,882 men aged 55 years or older were randomized to treatment with finasteride (5 mg/d) or placebo for 7 years, finasteride appeared to prevent or delay onset of prostate cancer.⁴⁴ Prostate cancer was detected in 803 (18.4%) of the 4368 men in the finasteride group and 1147 (24.4%) of the 4692 men in the placebo group. Over the 7-year period, the reduction in prevalence of prostate cancer with finasteride relative to placebo was 24.8%. Whether the 1-mg dose of finasteride used in the treatment of male pattern baldness has a similar effect is unknown. Also, the degree to which the results of the study can be generalized to the male population at large has not been determined.

SURGERY

Hair Transplantation. Surgical techniques, primarily hair transplantation, have been used for nearly half a century to manage male pattern baldness. Hair restoration surgical techniques and technology have advanced particularly rapidly over the past 2 decades. Because of these advances, results from surgical hair restoration appear much more natural and pleasing than in past decades; visual evidence that a surgical procedure was performed is not apparent to the casual observer. In contrast, surgical procedures in previous years could not provide these same advantages; today, unnatural results remain in patients who underwent procedures performed with older techniques. These patients often bear the physical and emotional scars of unnatural hair appearance and distrust the field of hair restoration surgery. The challenge for the hair restoration surgeon is to provide a level of expertise and honesty to these patients to restore their appearance and self-confidence. Accurate assessment of the hair restoration problem is paramount to designing the best possible solution for the patient. It is important to determine the problems encoun-

tered in attempting to achieve the objective in the previous procedures and to integrate the chief concerns of the patient into the current surgical plan.

Hair transplantation entails the transfer of hair follicles from the back or sides of the head, where hair grows independently of androgens, to balding areas.³ Many patients who undergo hair transplantation can receive a sufficiently dense transplant in 1 or 2 surgical sessions. Extremely natural and aesthetically pleasing results can be achieved with today's techniques.^{44,45} Results are approximately 70% evident by 5 to 6 months after surgery and can vary, depending on the extensiveness of presurgical hair loss, the density of transplanted grafts per unit area, patient expectations, and the surgeon's skill. Limitations of surgical approaches include cost and scarring.^{3,5} A density exceeding 40 follicular units/cm² in the donor area typically is required. Continued hair loss after surgery is normal because of the progressive nature of hair loss. The need for repeated surgeries is common, especially in transplantation in the crown area. To optimize results, pharmacotherapy is recommended in conjunction with hair transplantation to maintain the resident non-transplanted hair.

Scalp Reduction. In scalp reduction, areas of balding scalp are removed to bring areas of hair-containing scalp closer to one another. A valuable tool in some cases, scalp reduction is performed with limited frequency. Limitations of scalp reduction include potential widening of scars over time and the need for repeated scalp reductions because of the progressive nature of hair loss. However, in a carefully selected subset of patients with androgenetic alopecia, scalp reduction surgery can be beneficial.

COSMETIC AIDS

Cosmetic aids including wigs and hairpieces to hide hair loss can be used alone or in conjunction with surgery or pharmacotherapy. Wigs and hair systems today appear much more natural and weigh much less than in the past. Pigmented powders, lotions, and hair sprays can be used to assist in camouflaging hair loss by adhering to existing hair to create the illusion of increased density or by decreasing the color contrast between the hair and the underlying scalp. However, they do not treat the underlying condition.

DEFINING AND MANAGING PATIENT EXPECTATIONS

Regardless of which treatment modality is chosen for male pattern baldness, defining and managing the patient's expectations regarding therapy is paramount in determining outcomes. When consulted by an individual concerned

about male pattern baldness, it is important to determine what bothers the patient most about his hair loss. Determining the answer to this simple question can be most important in managing expectations and providing appropriate recommendations. Although current treatment modalities confer measurable hair growth, robust patient satisfaction, and improvement in body image in many patients, no treatment is successful in all patients. Furthermore, the degree to which objective improvement in hair growth correlates with satisfaction with the outcome of treatment can vary markedly from patient to patient. In this context, it is important that patients are accurately informed about the possible shortcomings and disadvantages as well as the potential promise of various treatment modalities, that their expectations of treatment are understood before treatment is initiated, and that those whose expectations are determined prospectively to be unrealizable with currently available interventions are not treated. Pretreatment and posttreatment photographs can be invaluable.

CONCLUSIONS

Generalist clinicians are called on increasingly to manage male pattern baldness, a pervasive condition that can be physically and psychologically harmful. Treatment modalities include pharmacotherapy, surgery, and cosmetic aids. Two US Food and Drug Administration–approved hair-loss pharmacotherapies—the potassium channel opener minoxidil and the DHT synthesis inhibitor finasteride—are safe and effective for controlling male pattern baldness with long-term daily use. Modern follicle-based hair transplants appear much more natural and more successfully meet patient objectives than older techniques. Promising future developments include pharmacotherapy with dutasteride (a DHT synthesis inhibitor like finasteride), hair follicle transplantation from nonautologous donors, and hair follicle cultures. Regardless of treatment modality, defining and managing patient expectations regarding therapy are paramount to determining outcome.

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