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Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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DUTASTERIDE AS AN EFFECTIVE TREATMENT IN ANDROGENETIC ALOPECIA – A LITERATURE REVIEW

Dawid Sewruk *(Corresponding Author, Email: dawidsew321@gmail.com)*
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Lublin, Poland
ORCID ID: 0009-0008-4153-7126

Karol Kanon
University Clinical Centre in Gdańsk, Gdańsk, Poland
ORCID ID: 0000-0001-6705-1302

Mathias Spitaleri
7th Naval Hospital in Gdańsk, Gdańsk, Poland
ORCID ID: 0009-0007-0293-1764

Wiktor Gąska
University Clinical Centre in Gdańsk, Gdańsk, Poland
ORCID ID: 0009-0003-8818-988X

Oskar Sienkiel
7th Naval Hospital in Gdańsk, Gdańsk, Poland
ORCID ID: 0009-0002-4524-0721

Filip Kieloch
1st Clinical University Hospital in Lublin, Lublin, Poland
ORCID ID: 0009-0003-5116-9703

Agnieszka Fitas
4th Clinical University Hospital in Lublin, Lublin, Poland
ORCID ID: 0009-0005-9285-9174

Wojciech Gąska
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Lublin, Poland
ORCID ID: 0009-0005-7621-3533

Karolina Dębek-Kalinowska
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Lublin, Poland
ORCID ID: 0000-0001-9931-6002

Julia Głowacka
Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Lublin, Poland
ORCID ID: 0009-0004-3262-5598

ABSTRACT

Introduction: Androgenetic alopecia (AGA) is a common hereditary condition characterized by progressive hair loss in both men and women. While not a direct health threat, AGA significantly affects psychological well-being due to the role of hair in self-image and perceived attractiveness. Dutasteride, initially approved for benign prostatic hyperplasia, has demonstrated greater efficacy than finasteride in treating AGA. Its use in mesotherapy may help reduce systemic side effects, although more research is needed to confirm safety and define optimal treatment protocols.
Aim of study: To review current evidence on the efficacy and safety of dutasteride in the treatment of AGA and assess its potential as a therapeutic option.
Methods and material: A literature search was conducted using PubMed and Scopus to identify relevant studies on the use of dutasteride in AGA.
Results: Dutasteride 0.5 mg has shown superior efficacy compared to finasteride 1 mg and placebo in increasing hair count and shaft thickness in men with AGA. As a dual 5α-reductase inhibitor, dutasteride offers broader inhibition than finasteride, which targets only the type 2 isoenzyme. Higher doses, such as 2.5 mg, may yield even greater improvements. Mesotherapy with dutasteride appears to be well tolerated and may reduce systemic exposure, though evidence remains limited, and further studies are required to determine long-term safety and appropriate dosing.
Conclusion: Although not officially approved for AGA, dutasteride demonstrates superior clinical efficacy compared to finasteride. Side effects, particularly regarding sexual function, appear similar; however, concerns about long-term risks such as depression and metabolic changes persist. These should be carefully considered in patient consultations. Mesotherapy may offer a safer delivery method, but more robust data are needed to support its widespread use.

KEYWORDS

Dutasteride, Androgenetic Alopecia, 5-Alfa-Reductase, 5aRi, Finasteride, AGA, Male Pattern Hair Loss

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Introduction

Androgenetic alopecia (AGA), also known as androgenic alopecia or male pattern hair loss (MPHL), is a hereditary disease that leads to progressive hair thinning and loss [1]. This predominantly affects the crown area of the scalp [2]. In this condition there are many factors involved such as genetic abnormalities, microinflammation, testosterone and its methabolism [3,4].The disease can affect both women and men[5,6].

Research shows that androgenetic alopecia has a negative psychological impact on both men and women, since hair is often viewed as an important external facial feature, even though the condition itself is not harmful from a medical standpoint [7]. This impact is largely due to the cultural importance of hair, as it is often associated with attractiveness, youth, and vitality in many societies and as a result, the high value placed on maintaining a full head of hair has driven the demand for products designed to prevent hair loss [7].

Androgenetic alopecia is among the most frequently encountered chronic conditions in dermatology clinics: this age-related disorder affects over 80% of Caucasian men and 42% of Caucasian women by the age of 70 [8]. AGA is marked by gradual hair loss, mainly in the central scalp area, though the pattern can vary. While it is most common in older adults, AGA can even begin as early as puberty [8].

State of knowledge

The human hair growth cycle spans 5 to 7 years and is characterized by asynchronous and stochastic phases, including anagen, catagen, and telogen [9]. Anagen is the active growth phase within the follicle, catagen is the transitional phase marked by cell apoptosis and cessation of growth, and telogen is the resting phase where the hair prepares to be shed, followed by the initiation of a new growth cycle through neogenesis [9]. Androgenic alopecia is characterized by the gradual shrinking of hair follicles, which causes terminal hairs

to transform into vellus hairs due to a disruption in the hair growth cycle, where the anagen phase (growth phase) shortens over time, while the telogen phase (resting phase) lengthens [1].

Dutasteride is an effective drug for decreasing prostate volume, alleviating lower urinary tract symptoms, improving urinary flow rates, and reducing the incidence of acute urinary retention and the need for surgical intervention. [10]. Dutasteride inhibits both type I and II 5-alpha-reductase, leading to a more significant reduction in serum DHT by over 90%, while finasteride, a selective inhibitor of the type II 5aR isoenzyme, reduces circulating DHT levels by only 70%, with the remaining DHT likely produced by type I 5aR [11]. Administration of oral dutasteride at a dose of 0.5 mg per day already results in a reduction of serum dihydrotestosterone levels by over 90% and an increase in serum testosterone levels [12].

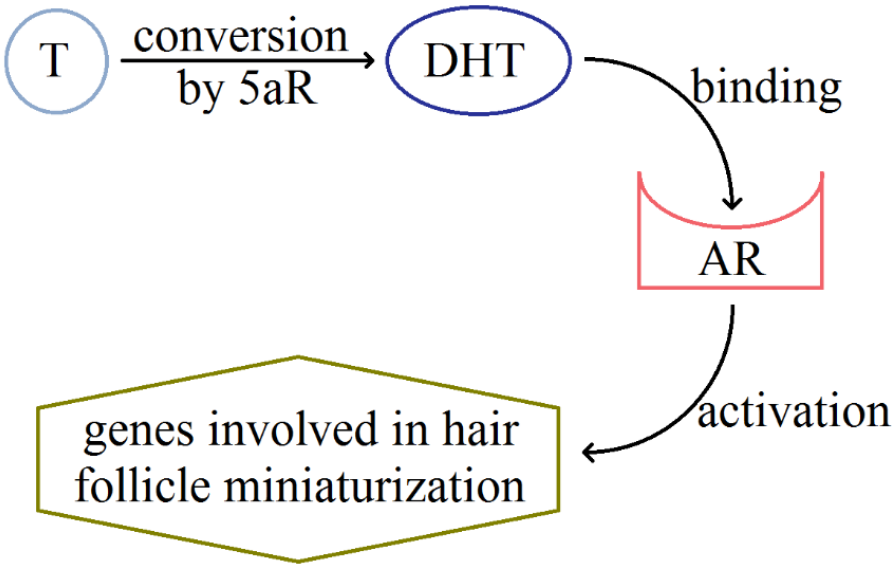

Fig. 1. *The influence of androgens on hair follicles.*

Pathogenesis of AGA

While the role of androgens in androgenetic alopecia is well recognized, the genetic inheritance pattern remains debated: the notion that a single autosomal gene accounts for this predisposition is commonly cited [3]. However, it seems more probable that the predisposition is influenced by multiple genes in a polygenic manner, with modulation by the androgen/estrogen ratio [3]. There is a possibility that part of this polygenic predisposition is inherited from father to son, potentially involving the Y chromosome or genes subject to parental imprinting [3]. Twin studies indicate high concordance rates (80-90%) in identical twins, and family history analyses revealing a significantly higher risk for men with a bald father, while the risk decreases if the father is not bald, although a positive family history on the mother's side or maternal grandfather's side also raises the risk.

Recent independent studies have increasingly highlighted the role of different inflammatory activators causing microinflamation in the development of androgenetic alopecia [4]. Evidence suggests that fibroplasia of the dermal sheath, which encases the hair follicle, may be a key terminal event leading to the miniaturization and regression of the pilosebaceous unit in AGA [4].

The primary pathological mechanism in androgenetic alopecia involves the conversion of testosterone to dihydrotestosterone (DHT) via the enzyme 5-α-reductase (5aR), which exists in three isoenzymes: type I, found mostly in hair follicles, sweat, and sebaceous glands; type II, predominantly located in the male genitalia, including the prostate, as well as the inner root sheath of hair follicles; and type III, which is distributed across the dermis, epidermis, mammary glands, and brain, though its exact role remains unclear [13]. DHT within cells acts as a more potent androgenic agonist compared to testosterone (T) [14]. DHT binds to androgen receptors (AR), causing a conformational change that leads to AR dissociating from heat shock proteins, translocating to the nucleus, and forming a homodimer that binds to androgen response elements (AREs) in the promoter regions of target genes, thereby initiating a genes activation process that progressively miniaturizes large, terminal hair follicles, resulting in shorter anagen phases, smaller hair matrices, and ultimately the production of vellus hairs, a hallmark of AGA [1,13].

Current pharmacological treatments of AGA

Hair transplantation is one of the therapeutic options for androgenetic alopecia, involving the autologous transplantation of hair [15]. In this surgical procedure hair follicles are being redistributed to restore hair density in areas impacted by AGA after excising a strip from the mid-occipital scalp followed by its stereomicroscopic dissection into follicular units (wich results in long thin scars) or after removing individual follicular units from the donor hair using 0.8-1.0 mm punches (wich is now the most common donor harvesting technique becouse of better cosmetic effect on donor area) [15].

The only FDA-approved medications for treating AGA are minoxidil, a potassium channel opener, and finasteride, a DHT synthesis inhibitor, both of which are effective with consistent, long-term use [1]. Medical therapy is most beneficial when initiated early in hair loss and can be used as a standalone treatment, though it is also crucial in surgical interventions to preserve surrounding native hair and improve overall aesthetic outcomes [1].

Initially introduced in the early 1970s as a treatment for hypertension, minoxidil's side effect of hypertrichosis, including hair regrowth in male balding, led to the discovery of its direct stimulatory effect on dermal papillae or follicular hair matrix cells, which in turn resulted in the development of a topical formulation for treating androgenetic alopecia in both men and women [16].

Finasteride, approved by the US FDA in 1997, is currently the only oral medication approved for treating male androgenetic alopecia (MAGA); it is a type 2, 5-alpha reductase inhibitor that prevents the conversion of testosterone to dihydrotestosterone (DHT) [8]. Administration of finasteride results in about 70% reduction in DHT serum levels [17]. Moderate-quality evidence suggests that long-term use of finasteride at 1 mg daily improves hair growth in about 30% of patients, particularly in the vertex area, though it is associated with an increased risk of erectile dysfunction, estimated at an absolute increase of approximately 1.5% compared to placebo [8].

Dutasteride - an off label treatment for AGA

Dutasteride is used as a off-label treatment of androgenic alopecia [18]. Despite lacking FDA approval, randomized clinical trials have provided substantial evidence of dutasteride's effectiveness in treating mild to moderate androgenetic alopecia in men, demonstrating its efficacy both relative to placebo and in comparison to finasteride [19] [20][21].

Effectiveness and safety of dutasteride

The study demonstrated that dutasteride 0.5 mg is more effective than finasteride 1 mg and placebo in increasing hair count and width after 24 weeks of treatment in men with androgenetic alopecia, while showing no significant differences in the incidence of adverse events among the treatment groups [22].

Another study confirmed that 0.5 mg of dutasteride is more effective than a placebo and well tolerated, with a safety profile comparable to that of the placebo group, as the differences in the frequency of total and drug-related adverse events were not statistically significant, with most adverse events being mild in nature; furthermore, no significant differences were observed between the groups regarding sexual function [20].

Finasteride exhibits a mean terminal elimination half-life of approximately 7 hours, with a shorter duration observed in younger male individuals and a prolonged half-life typically seen in men aged over 70 years [23]. Dutasteride has a serum half-life of approximately 4 weeks, as demonstrated by the sustained suppression of DHT even after discontinuation of treatment with doses of 0.5 mg and 2.5 mg [21].

Dutasteride at a 2.5 mg dose, a dual 5α-reductase inhibitor, enhanced hair growth in balding men more rapidly and to a greater extent than finasteride, a selective type 2 inhibitor, and was generally well tolerated, demonstrating the significant synergistic effect of inhibiting both type 1 and type 2 5α-reductase in the treatment of male pattern hair loss [21].

A meta-analysis comparing efficacy and safety of dutasteride and finasteride provides us with information that dutasteride appears to be more effective than finasteride in treating AGA, with both drugs exhibiting comparable rates of adverse effects, particularly regarding sexual dysfunction [24].

In a unique study involving male identical twins with androgenetic alopecia, treatment with dutasteride was shown to slow hair loss progression and promote hair growth, whereas the placebo group exhibited disease progression [25]. Since male pattern hair loss is genetically controlled and identical twins share the same genetic code, comparing paired data between twins offers a highly efficient method for evaluating treatment efficacy in a limited patient population, as each twin acts as a control [25].

Another interesting study reports the case of a 46-year-old woman with androgenic alopecia unresponsive to minoxidil, who initially showed some improvement with finasteride; however, due to limited progress and ongoing severe psychological distress from androgenic alopecia, a second 5α-reductase inhibitor, dutasteride, was introduced, with efficacy assessed through clinical evaluation, trichogram, and monitoring of mean hair diameter via computer dermoscopy; after 6 months of therapy, significant improvement was observed, and by 9 months, the clinical diagnosis of androgenic alopecia was no longer applicable, with no side effects reported [26].

The approved dose of dutasteride for benign prostatic hyperplasia is 0.5 mg daily, with limited data on the safety of higher doses; it is not approved for MPHL treatment, where benefits must be weighed against potential side effects like gynecomastia, reduced sperm count, and interactions with CYP 3A4 inhibitors [21].

One study observed a link between the use of dutasteride and the occurrence of depression, while no such association was found with finasteride; in terms of severity, 5ARIs primarily caused mild rather than moderate or severe depression [27].

Given that finasteride and dutasteride are frequently prescribed for long-term treatment of male pattern hair loss, it is postulated that men using these drugs may be in a state of androgen deficiency, placing them at increased risk for non-alcoholic fatty liver disease, insulin resistance, type 2 diabetes, dry eye disease, potential kidney dysfunction, and other metabolic disorders [28].

The extended half-life of dutasteride compared to finasteride implies that, although not permanent, the impact on sexual function or spermatogenesis may be more pronounced and enduring, potentially lasting for weeks rather than just days [29].

Dutasteride via mesotherapy

Mesotherapy involves the administration of pharmacological agents via intradermal injections targeted at a specific anatomical area, typically resulting in minimal or absent systemic effects [30]. Administration of dutasteride via mesotherapy is an effective way to treat androgenic alopecia and it may reduce the incidence of side effects due to the localized action of the medication, but more research is needed to confirm this, as well as to determine the appropriate dosage and frequency of injections [31]. It is important to note that systemic absorption may still occur and could negatively impact spermatogenesis and potentially lead to other side effects [31]. The authors of study about mesotherapy using dutasteride claim that their patients did not experience decreased libido, erectile or ejaculatory dysfunction and the only reported adverse effects were pain during the injection, mild headache, or tension lasting one or two days [32]. Another study suggests that dutasteride mesotherapy administered only once every three months yields positive results, with no observed side effects and no changes in blood DHT levels; however, the study was conducted on a small group of patients [33].

Conclusions

Based on the literature review, it can be concluded that although dutasteride is not officially approved for the treatment of androgenetic alopecia, it demonstrates higher efficacy than finasteride in increasing hair density and thickness. Despite comparable rates of side effects, particularly regarding sexual dysfunction, there are concerns about the long-term use of these drugs, including the potential risk of depression and other metabolic disorders. This is an issue that should be carefully considered in consultation with the patient, taking into account the impact of the disease as well as the potential side effects on the patient's quality of life. It should be also noted that dutasteride, when used in mesotherapy, may offer benefits by reducing the incidence of side effects, but further research is needed to evaluate its safety and optimal dosing regimens.

Authors contribution:

Conceptualization: Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Methodology: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka

Software: Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Check: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka

Formal Analysis: Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Investigation: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka

Resources: Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Data curation: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka

Writing – rough preparation: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka, Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Writing – review and editing: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka, Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Visualization: Filip Kieloch, Wiktor Gąska, Agnieszka Fitas, Karol Kanon, Julia Głowacka

Supervision: Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

Project administration: Dawid Sewruk, Mathias Spitaleri, Oskar Sienkiel, Wojciech Gąska, Karolina Dębek-Kalinowska

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