ŁOPUSZYŃSKA, Anna and OCHYRA, Łukasz. The latest reports on androgenetic alopecia. Quality in Sport. 2024;18:53366. eISSN 2450-3118.
https://dx.doi.org/10.12775/QS.2024.18.53484
https://apcz.umk.pl/QS/article/view/53484

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 16.07.2024. Revised: 1660.08.2024. Accepted: 05.08.2024. Published: 12.08.2024.

The latest reports on androgenetic alopecia

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Abstract

Androgenetic alopecia (AGA) is the most common type of hair loss among women and men. AGA is caused by a combination of genetic factors resulting in sensitivity to dihydrotestosterone (DHT), which binds to the androgen receptor (AR), mediating a series of biomolecular changes that lead to changes in the hair. There are many mechanisms that can lead to AGA, some of which remain undiscovered. Treatment of AGA is also important for psychological reasons, as it constitutes a significant psychosocial stressor in the lives of people affected by this disease and worsens the quality of life. The only medications approved for the treatment of AGA are oral finasteride and topical minoxidil. These drugs may be associated with side effects, hence the need to look for other treatment methods, especially for patients who cannot tolerate approved methods. The current review focuses on the latest reports on the pathogenesis and treatment of androgenetic alopecia.

K**eywords:** androgenetic alopecia, alopecia, dermatology

Introduction

Androgenetic alopecia (AGA) is the most common type of hair loss and is characterized by progressive miniaturization of hair follicles [1]. AGA affects not less than 50% of men under 50 years of age and 70% of men over 70 years of age, and in the case of women 6% and 30-40%, respectively. Men's androgenetic alopecia is characterized by baldness on the top of the head and a shift of the frontal hairline to the rear. In women, there are three AGA patterns: Hamilton type - "male" type, Ludwig type - central diffuse thinning in the frontal area and Olsen type - "wide part" type [2]. The severity of AGA is commonly assessed using the Hamilton-Norwood scale (HNS) in men and the Ludwig classification in women [3]. It can be seen in the trichoscopic images increase in the number of yellow dots, pilosebaceous units with only one hair and perifollicular discoloration. The percentage of thin hair (<0.03 mm) in AGA is significantly higher than in healthy people [4]. It has been suggested that AGA may be an indicator of metabolic syndrome (MetS) and cardiovascular disease (CVD), as well as a marker of early atherosclerosis [5].

Pathogenesis

Androgenic alopecia is caused by a combination of genetic factors resulting in sensitivity to dihydrotestosterone (DHT), which binds to the androgen receptor (AR), mediating a series of biomolecular changes leading to hair changes [6].Dermal papilla cells in the balding scalp of AGA patients show signs of aging, such as loss of replicative potential, changes in cell size and shape, and reduction or loss of characteristic molecular markers/signature [7]. Recent reports suggest that miR-221, which is located in the same part of the hair follicle as AR, significantly inhibited hair growth and proliferation of dermal papilla and dermal sheath cells. AR has been shown to promote miR-221 transcription, which suppresses the expression of IGF-1, which plays a key role in regulating the hair cycle. This provides an opportunity for a new biomarker and a potential therapeutic target in the treatment of AGA [8]. Z. Deng et al reported that paracrine signaling via AR, mainly TGF-β from dermal papilla cells induces apoptosis of vascular endothelial cells. Vascular regression at the early stage of miniaturization of hair follicles in the development of AGA confirms the need to start treatment of alopecia as early as possible [9]. While various growth factors and signaling pathways are involved in the hair cycle process, activation of Wnt/β-catenin signaling plays a key role in hair follicle regeneration [10]. CXXC5 has been reported to mediate hair loss through the DHT-PGD_2 by suppressing Wnt/β-catenin signaling [11]. Q. Liu et al report that genes related to the HIF-1 pathway (EGLN1, EGLN3) and Wnt pathway inhibitors (SERPINF1, SFRP92) may play an important role in AGA, which may constitute a therapeutic target [12].

Treatment

There are many pathogenic mechanisms associated with the development of AGA, and we do not know how many are yet undiscovered. Hence, a multi-therapeutic approach is needed. Drugs that can regulate hair follicle cycling, protect follicle cells from autoimmune inflammatory cell infiltration, and control the expression of AR and related enzymes represent an important therapeutic approach [13]. Treatment of AGA is also important for psychological reasons, as it constitutes a significant psychosocial stressor in the lives of people affected by this disease and worsens the quality of life [14]. However, there are still many unknowns in our knowledge on this topic, hence many different substances, drugs, and administration methods are being investigated for the treatment of AGA. The only drugs that are approved for the treatment of AGA in men are oral finasteride and topical minoxidil. For women, topical minoxidil is the drug of first choice [15].

Finasteride is a synthetic 5α-reductase inhibitor that inhibits the conversion of testosterone to DHT. In men, it is used at a dose of 1.0 mg/day, in postmenopausal women 2.5-5.0 mg/day, off-label [2]. In studies comparing it at a dose of 5 mg/day with other possible therapies, the greatest increase in total hair number was observed after 48 weeks. This drug was significantly more effective than 2% topical minoxidil (mean difference, 20.7 hairs/cm2; 95% CI, 9.5-31.9 hairs/cm2). The greatest increase in final hair number after 48 weeks occurred after administration of finasteride at a dose of 1 mg/day, which was significantly more effective than topically applied minoxidil 2% and 5% [16]. The treatment is effective, but side effects are a serious problem. Post-finasteride syndrome (PFS) is a set of serious adverse reactions manifested by clinical symptoms that develop and persist in patients during and/or after treatment with finasteride or dutasteride in men. These serious side effects include permanent or irreversible sexual, neurological, physical and psychological side effects [17]. In studies on 1 mg/day finasteride or 0.5 mg/day dutasteride, an increased risk of sexual dysfunction was observed in men (in the case of dutasteride, this risk was not statistically significant) [18].

For postmenopausal women, it has been noted that 5 mg of finasteride administered orally daily may be an effective and safe treatment option, especially when combined with other medications such as topical estradiol and minoxidil. Topical finasteride has also been found to be more effective than other topical preparations in the treatment of hair loss [19]. High doses of finasteride caused side effects analogous to men. However, no systemic side effects of topical finasteride have been reported in patients with FPHL (female pattern baldness), except for a decrease in serum DHT concentrations. Minimal local side effects such as pruritus and irritation were reported but were perfectly tolerated [20].

Topical finasteride (0.25% solution applied once daily in a volume of 50 µl to 200 µl) significantly improves hair number compared to placebo and is well tolerated. Its effect is similar to oral finasteride, but with much lower systemic exposure and less impact on serum DHT concentrations [21].

Dutasteride is a second generation 5α-reductase inhibitor, it is more potent than finasteride due to its ability to inhibit both type I and type II isoenzymes, thus leading to a 90% reduction in serum DHT levels, while finasteride only reduces by 70% [22]. The greatest increase in total hair number after 24 weeks occurred after administration of dutasteride at a dose of 0.5 mg/d, which was significantly more effective than finasteride and minoxidil [16]. Other forms of administering this drug are also being investigated. Dutasteride microinjections appear to be a safe and potentially effective alternative in the treatment of AGA, which is likely to be

increasingly used in the future, especially in patients who do not want to undergo systemic treatment or as an adjuvant therapy [23]. Dutasteride appears to provide superior efficacy in the treatment of AGA compared to finasteride. Most likely, both drugs have a similar rate of side effects, especially in the case of sexual disorders [24].

Minoxidil, initially introduced as an antihypertensive drug, is currently one of the basic drugs used topically in AGA. It is most often used in concentrations of 2% and 5% [25]. When administered orally at a dose of 5 mg/d, the greatest increase in final hair count was observed after 24 weeks, which was significantly more effective than the 0.25 mg/d dose of minoxidil (mean difference 43.6 hairs/cm2; 95% CI, 29.7 -57.7 hairs/cm2) and local forms - 2% [mean difference, 29.3 hairs/cm2; 95% CI, 21.1-37.5 hairs/cm2] and 5% [mean difference, 29.8 hairs/cm2; 95 % CI, 19.7–39.8 hairs/cm2]. Minoxidil at a dose of 5 mg/day was significantly more effective than finasteride at a dose of 1 mg/day (mean difference, 10.4 hairs/cm2; 95% CI, 2.2–18.6 hairs/cm2) [16]. Minoxidil has been reported to act not only on AR but also on steroid 17-alpha-hydroxylase/17,20 lyase (CYP17A1) and aromatase (CYP19A1). Inhibiting the expression of AR and CYP17A1, as well as increasing the activity of CYP19A1, reduces the formation and binding of DHT and supports the production of estradiol [26].

Cetirizine is a second-generation antihistamine, derived from the metabolism of hydroxyzine, highly specific for H1 receptors and having antiallergic properties [27]. Cetirizine inhibits the release of prostaglandin D2, which inhibits hair follicles, and stimulates the release of prostaglandin E2, which stimulates hair follicles. In a study in men comparing 1% cetirizine solution and 5% topical minoxidil, it was noted that 1% cetirizine solution was effective in hair growth without any complications in the treatment of male AGA (although greater improvement was noticeable with minoxidil) [28]. A study was also conducted in women comparing the combination of cetirizine and minoxidil with topical minoxidil alone. In the cetirizine/minoxidil group, there was a statistically significant change from baseline in frontal and terminal hair density and vellus hair density ($P < 0.0005$) with significant increases in apical hair shaft thickness and mean number of hairs per follicular unit. However, side effects did not differ significantly between groups [29].

Platelet-rich plasma (PRP) has been described as a small volume of plasma containing higher concentrations of platelets than those found in peripheral blood. Initially, it was used as a transfusion product to treat thrombocytopenia. To date, it has been discovered that there are several growth factors and cytokines that can accelerate wound healing and tissue regeneration, leading to a wider range of applications in medicine, such as sports, regenerative and aesthetic medicine [30, 31]. More and more research is focusing on the use of PRP in

AGA therapy [32]. A positive effect of PRP on AGA was noted (including improvement in hair thickness and density), without major side effects, therefore it can be considered a safe and effective alternative hair loss treatment procedure compared to minoxidil and finasteride [33]. In a study in men, the combined use of PRP and minoxidil appears promising in the treatment of AGA. PRP increases the proliferative activity of HF cells and improves hair morphology in AGA patients [34]. The use of autologous PRP injections in women with AGA seems promising, providing more consistent results regarding final hair density [35]. PRP used in women has been shown to provide a high level of satisfaction and improve quality of life. In light of this evidence, PRP may be offered to patients who have not responded to or cannot tolerate topical minoxidil, as well as in combination with topical and oral treatments [36]. Additionally, combining PRP with laser therapy, microneedling, dermal fillers, and autologous fat grafting produces synergistic effects, leading to improved aesthetic results. Future studies should standardize PRP treatment protocols [37].

Other methods

Botulinum toxin is an endotoxin produced by the anaerobic bacterium Clostridium botulinum. This endotoxin is a dimeric protein that acts at the neuromuscular junction to block the release of acetylcholine, thereby reducing motor unit contraction [38, 39]. In the case of AGA treatment, botulinum toxin type A is a safe and effective method, hair growth and density have been noticed, and the area of hair loss is reduced. Moreover, in combination with oral finasteride, an even better therapeutic effect was observed [40].

Low-level light therapy (LLLT) causes the photons that are emitted to oxidize cytochrome C oxidase, thereby activating the electron transport chain and increasing ATP production. Endogenous growth factors and nitric oxide are increased, leading to cell proliferation and vasodilation. In the treatment of AGA, we use LLLT 650-900 nm and a power of 5 mW [41]. Studies have reported that LLLT is an effective, well-tolerated treatment method, with a good safety profile, good synergistic effect with other products and few mild side effects [42].

Stem cells are unspecialized cells of the human body. They can differentiate into any cell of the body [43]. Stem cells have self-renewal, migration, anti-inflammatory and immunomodulatory functions that are necessary for the repair and regeneration of damaged tissues or organs [44]. Y. Mao et al report that available experimental studies have shown that both stem cells and non-cell therapies based on stem cells can support hair regeneration and prevent hair loss [45]. Patients with alopecia have not reported any serious side effects during and after stem cell treatment. The use of stem cells in the treatment of AGA seems to be a

promising alternative to standard treatment or could act as a complementary therapy, improving the effect of primary treatment [46].

In the study conducted on mice, cell-free fatty extract (CEFFE) was used. An increase in the rate of hair transition to anagen and an increase in the percentage of hair coverage were observed. Moreover, the number of CD31+ capillaries and Ki67+ cells increased, suggesting dermal papilla cell proliferation, modulated cell cycle arrest, inhibited DHT-induced apoptosis, reduced intracellular DHT concentration in DPCs, and downregulated AR expression [47].

In vivo studies in men and women with AGA confirmed that the use of topical preparations containing caffeine reduces hair loss. The results obtained so far have shown that caffeine-containing hair loss therapies can be as effective as drug-based therapies, while still providing the good safety profile commonly associated with cosmetic products [48].

An oral supplement containing hydrolyzed fish collagen, taurine, cysteine, methionine, iron, and selenium has been shown to improve the clinical effectiveness of specific hair loss therapies in people with AGA [49].

Conclusions

The treatment of androgenic alopecia is challenging and a chronic problem for patients. Drugs approved for the treatment of AGA, i.e. topical minoxidil and oral finasteride, although effective, may be associated with side effects. The other therapeutic options described in the above review are promising and provide hope for patients who cannot tolerate primary treatment. Further large sample size, double-blind, randomized, placebo-controlled studies are needed.

Author's contribution

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All authors have read and agreed with the published version of the manuscript.

Funding Statement

The study did not receive special funding

Informed Consent Statement

Not applicable

Acknowledgments

Not applicable

Conflict of interests

Authors have declared no conflict of interests

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