Update Treatment of Male Androgenetic Alopecia

Damai Trilisnawati, Sarah Diba, Yuli Kurniawati, Suroso Adi Nugroho, Rusmawardiana, Raden Pamudji

Department of Dermatology and Venereology, Medical Faculty of Sriwijaya University/Dr. Mohammad Hoesin General Hospital, Palembang

ABSTRACT

Background: Male androgenetic alopecia (MAGA), also known as androgenetic alopecia, is the most common hair loss in males who have a genetic predisposition. The pattern of baldness in MAGA starts from the frontal area in a triangular pattern, followed by progressive thinning of the vertex until baldness occurs. Generally, the diagnosis of MAGA is established by clinical examination. FDA has approved a combination of topical minoxidil and oral finasteride for MAGA treatment. Currently, there is another treatment option like dutasteride, a prostaglandin analog, ketoconazole, and co-adjuvant therapy like laser therapy, hair transplantation, and so on. **Purpose:** To provide an updated treatment for MAGA. **Review:** Etiopathogenesis of MAGA is influenced by genetic susceptibility and hormonal factors. The European Consensus Group set the evaluation diagnosis of MAGA to include a historyof hair fall, physical examination, hair examination, supporting examination, and clinical documentation. There are therapeutic options for MAGA, including antiandrogen therapies, androgen-independent therapies, and co-adjuvant therapies. The FDA has approved a combination of topical minoxidil and oral finasteride for MAGA treatment. MAGA may affect patients' quality of life and self-esteem. In general, patients expect higher. **Conclusion:** MAGA is the most common progressive hair loss in males. The MAGA therapy is expected to achieve cosmetically significant regrowth and to slow additional hair loss.

Keywords: male androgenetic alopecia, androgen, male hair-loss, minoxidil, oral finasteride.

Correspondence: Damai Trilisnawati, Department of Dermatology and Venereology Medical Faculty of Sriwijaya University / Dr. Mohammad Hoesin General Hospital Palembang, Jl. Jenderal Sudirman Km 3,5 Post code 30126, Palembang, South Sumatera, Indonesia. Phone number: +6282377474005.

BACKGROUND

Male androgenetic alopecia (MAGA) is the most common hair loss in males with a genetic predisposition. MAGA is characterized by miniaturization of nonscarring progressive hair follicles with shortening of the anagen phase, and it usually has specific distribution patterns.[1–3] Genetic susceptibility and hormonal affect the pathogenesis of MAGA. The incidence and severity of MAGA increase with age.[1,2] Androgenetic alopecia is more frequent in males than females. It is estimated that 50–60% of males experience MAGA at the age of 50, increasing to 80% at the age of 70.[1] The incidences of MAGA in Chinese, Japanese, and African males are lower than Caucasian males.[4]

In males, the pattern of baldness starts from the frontal area in a triangular pattern, followed by progressive thinning of the vertex until baldness occurs. Generally, the diagnosis of MAGA is established on a clinical basis.[1] There are several options for MAGA therapy, which are antiandrogen, androgenindependent, and co-adjuvant therapy.[5] FDA has approved a combination of topical minoxidil and oral finasteride for MAGA treatment.[1,5] This literature review discusses another therapy option other than finasteride and minoxidil. It is dutasteride, a prostaglandin analog, ketoconazole, and many co-adjuvant therapies like laser therapy, hair transplantation, and so on. Hopefully, this review can be part of the consideration for choosing a new better treatment.

REVIEW

In terms of anatomy, hair has three parts, namely the top (infundibulum), middle (isthmus), and bottom (inferior segment).[3] The interior of the hair bulb is invaginated at the base of the dermal papillae.[6] Hair grows with a continuous cyclic pattern in three phases, namely the anagen, catagen, and telogen that last for 2–6 years, 1–2 weeks, and 5–6 weeks, respectively, and they are exogenous. A normal hair cycle causes hair replacement on the scalp every 3–5 years.[3,6] Hair follicle growth stops and enters the physiological involution stage of transition or the catagen phase when active growth ends. In the catagen phase, the bulbar portion of the follicle is

degraded, but the hair follicle stem cells remain in the bulge. After that, the follicles undergo apoptosis and enter the telogen phase.[6]

Based on the pathogenesis of MAGA, androgens are the main hormones involved in the interaction of the papillae of the dermis and hair follicles in the miniaturization of hair follicles. Two androgen hormones involved are testosterone and 5α-dihydrotestosterone (DHT). Testosterone is converted to DHT by the 5α-reductase enzyme.[3] Hair growth is cyclic. In MAGA, the anagen duration decreases while the telogen duration remains constant and even lengthens. This causes a decrease in the ratio of anagen to telogen.[3,7] Changes in hair cycle dynamics in MAGA cause progressive and gradual miniaturization of hair follicles. Papillary dermis is very important for maintaining hair growth and being the target of androgen-mediated changes to the hair cycle and miniaturization of hair follicles. The mechanism of decreased size of hair follicles is still unclear, and it can be caused by cell apoptosis, decreased keratinocyte proliferation, loss of cell adhesion that causes dermal papilla fibroblasts to migrate to the dermis or migration of dermal papilla cells into the dermis sheath.[3] It is hypothesized that MAGA results from chronic scalp stretching during bone growth in puberty and post-puberty, and/or chronic contraction of muscles around the galeaaponeurotica (GA), fibrous membrane layers, and under the hair follicles.[8] There is oxidative stress in the dermal papilla of MAGA patients due to an increase in substrates that are susceptible to oxidative damage that can trigger an inflammatory response.[9] In MAGA, there is also an increase in sebum colonization of *Propionibacterium acnes* and porphyrins. *P. acnes* metabolizes sebum, producingporphyrins and proteins that play a role in inflammation through ROS and activation of proinflammatory cytokines, IL-1, and TNF-α.[8] The arrector pili muscle (APM) plays a role in maintaining hair follicle integrity. Torkamani et al. (2014) in a study examining the histopathology of APM in 13 hair loss patients, found that APM degeneration in MAGA was replaced by adipose tissue. APM attaches to the bulge, which is an important source of stem cells for hair cell regeneration. Signal interference is suspected to regulate stem cells or interfere with APM regeneration.[7,10]

Clinical manifestations of MAGA can be easily recognized because of the progression of patterned hair loss. The pattern of baldness starts in the frontal area. The hairline widens to form a characteristic description of the letter "M".[1] The main manifestation of MAGA is the decline in the frontal hairline crown baldness. The bald line meets and forms the hairline at the edges and back of the scalp. The progressions of MAGA is generally classified on the Hamilton-Norwood scale types I to VII.[11] MAGA diagnosis is issued by the European Consensus Group including history taking, physical examination, hair examination, supporting examination, and clinical documentation (Figure 1).[1]

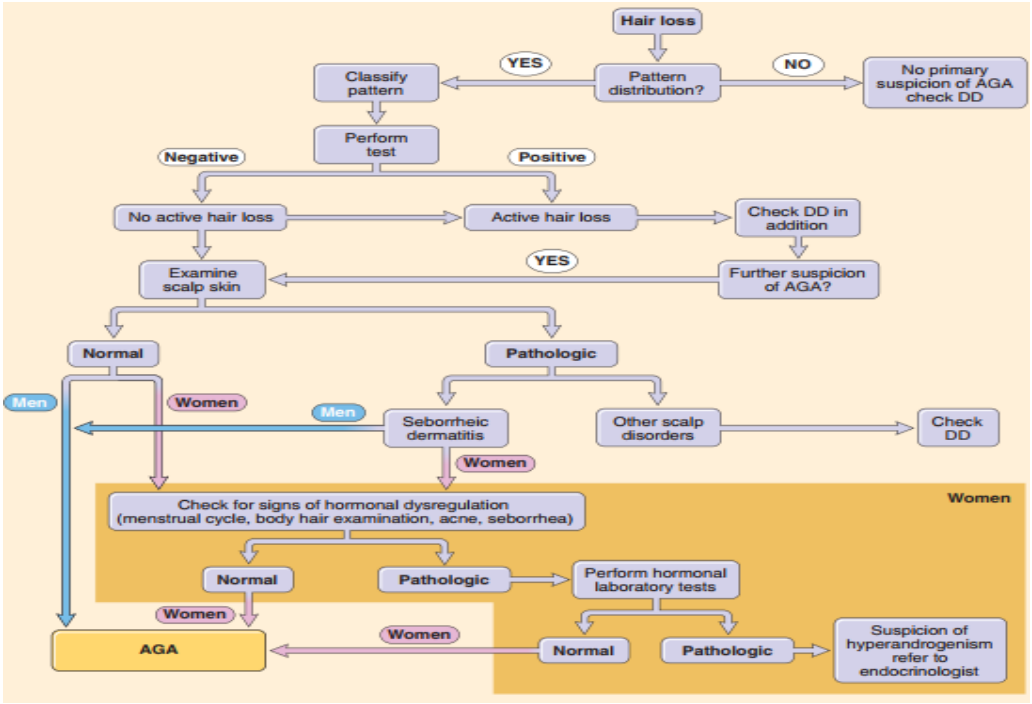

Figure 1. Male androgenetic alopecia (MAGA) diagnosis algorithm.[1]

The management of MAGA depends on various factors, including efficacy, practicality, risk, and cost. Some therapeutic modalities for MAGA consist of antiandrogen, androgen-independent, and co-adjuvant therapies.[12] Finasterid 1 mg and minoxidil 2–5% are drug choices approved by the United States Food and Drug Administration for MAGA.[13]

Finasterid is a type 2 5α-reductase inhibitor that reduces the conversion of testosterone to dihydrotestosterone (DHT), which plays a role in miniaturization of hair follicles. An oral dosage of 1 mg per day shows better results than minoxidil with effects of regrowth of hair. Finasteride decreases the median scalp DHT levels. The combination of oral finasteride with topical minoxidil is said to increase its therapeutic effect. Finasteride should be given for at least 6–12 months. After two years of therapy, two-thirds of patients experienced improvement. A 5-year trial showed fewer hair loss rates than untreated men. The therapeutic effect disappears within 12 months after therapy is stopped. Some studies suggest finasteride works by reactivating hypotrophic hair follicles by accelerating and extending the anagen phase, but does not convert velus hair into terminal hair.[5,14]

Table 1. First-line management for MAGA[5]

Medication	Treatment approval: US FDA	Mechanism of action	Dosage recommendations[a]	Major adverse effects
Finasteride	Approved	5α-reductase inhibitor	1 mg once daily	Sexual adverse effects
Dutasteride	Approved in several countries, e.g., Korea and Mexico	5α-reductase inhibitor	0.5 mg once daily	Sexual adverse effects
Topical finasteride	Not approved/off-label	5α-reductase inhibitor	1 % topical gel or 0.25 % topical solution application once daily to scalp	Systemic absorption leading to the same sexual adverse effects of oral finasteride is not clear yet
Minoxidil	Approved	Unknown, possible antiandrogenic, vasodilatory, and anti-inflammatory effects	5 % solution; topical application twice daily	Hypertrichosis Contact dermatitis
Latanoprost	Not approved/off-label	Prolongs the anagen phase	0.1 % solution; topical application once daily	Erythematous reaction
Ketoconazole	Not approved/off-label	Decreases hair follicle DHT levels	2 % shampoo; leave on for 5 min and then rinse; use 3 days a week	

DHT dihidrotestosterone, *FDA* Food and Drug Administration

[a] According to the main studies/reports about each medication

Male androgenetic alopecia (MAGA)

The safety of drug use showed thatfinasteride has no hepatotoxic or nephrotoxic effects. It is not recommended in patients with liver disease because the metabolic process occurs in the liver. Finasterid sexual side effects include decreased libido, erectile dysfunction, and decreased ejaculatory volume. Depression and suicide ideas have also been reported, especially in patients with permanent sexual dysfunction. Response to treatment must be assessed every six months, and in some male patients, the improvement is not observed before the 12th month. Therapy is continued to maintain effectiveness. In the case of 1 mg finasteride therapy for 12 months is not effective, another type of 5α-reductase inhibitors namely dutasteride, inhibiting type I and type II isoenzymes, a dose of 0.5 mg per day can be considered.[5]

Dutasteride is a 5α-reductase inhibitor that blocks the activity of type I and II isoenzymes and reduces serum DHT levels by 90% compared to finasteride, which is 70%. Dutasteride 0.5 mg is approved for BPH therapy in the world. However, it has only been approved for MAGA therapy in only a few countries, including Korea and Mexico. In clinical trial studies, there were no significant differences in side effects between treatment groups and placebo. A combination of type I and II 5α-reductase inhibitors is useful in the treatment of male hair loss.[5]

Minoxidil prevents disease progression and causes an increase in hair thickness, and thickness of hair strands in MAGA patients >18 years old (2–5% solution; 5% 1 ml twice per day). Treatment response must be assessed every six months. Therapy is continued to maintaineffectiveness. Patients should be informed about increased telogen hair loss at the beginning of 8 weeks of therapy.[1,14]

The mechanism of minoxidil in hair growth is still unclear but possibly mediated by opening of the potassium channel, causing an increase in skin blood flow and an increase in VEGF levels and promoters of hair growth in the dermal papillae. Several studies

have shown that minoxidil increases the production of prostaglandin E2 (PGE2) through stimulation of prostaglandin endoperoxide synthase-1 to increase hair growth.[5]

The most common side effects of minoxidil are contact dermatitis and facial hypertrichosis. Topical ingredients, especially propylene glycol, can cause skin irritation or allergies. A temporary increase in hair loss at the beginning of treatment was observed in some patients for 2 to 8 weeks after treatment initiation. This shows the efficacy of minoxidilas telogen follicles re-enter the anagen phase, which can last for several weeks. This temporary hair shedding resolved within several weeks during treatment.[5,15]

In 2008, the FDA approved bimatoprost, another PGF2 analog, for the treatment of eyelash hypotrichosis. This new research shows that prostaglandin F2 (PGF2) and its synthesis are expressed on the scalp of MAGA. In addition, research also shows that high PGF2 levels can induce miniaturization, oil gland hyperplasia, and alopecia in mice. The topical application of PGF2 also inhibits hair growth in mice. Research also shows that PGF2 levels, a promoter of hair growth, are higher in normal scalp than in MAGA scalp. This shows that the balance between various prostaglandins can control hair growth. In addition, studies have shown that the PGF2 receptor, GPCR44, is a potential target for treatment.[5]

Ketoconazole is an imidazole class antifungal. The mechanism of action is still unclear, but a study supports the hypothesis that 2% ketoconazole shampoo interferes with the DHT pathway. Oral finasteride combination ketoconazole shampoo has been clinically proven effective in the treatment of MAGA.[7]

Follicular unit transplantation is considered a standard gold technique. A small follicle unit can give better results. Patients should be informed that postoperative telogen effluvium may occur for a while. Complications such as infection, pain, and growth failure of transplanted hair are rare.[5] Patients who do not meet the hair transplant requirements can use a camouflage method such as micropigmentation, which is recently developed. Hair and wig extensions are also included.[5]

Platelet-Rich Plasma (PRP) is a plasma preparation with a high platelet concentration. The use of PRP for hair loss has been studied in several studies. Platelets contain growth factors involved in the phase of hair growth. Several growth factors play a role in the growth and maintenance of follicles such as platelet-derived growth factor (PDGF) to stimulate stem cell mitosis, transforming growth factor-b to activate dermal papillae and inhibit apoptosis during the cell cycle, vascular endothelial growth factor (VEGF) helps form microcirculation.[5] Singhal *et al.* observed an increase in hair growth in MAGA patients after injection of autologous PRP.[16] PRP was first used for hair transplants, increasing hair follicle growth, increasing hair density and amount, and accelerating the time of hair formation. The disadvantages of PRP is pain during the procedure and possible non-permanent effects.[5,12]

Scalp micro needling was first reported in 2012. Two studies have shown an increase in gene expression related to hair after micro needling in mice. Possible mechanisms are (a) the release of platelet-derived growth factor (PDGF) through platelet activation and the mechanism of wound regeneration; (b) activation of follicle stem cells during wound healing; and (c) gene overexpression associated with hair growth such as VEGF, b-catenin, Wnt3a, and Wnt10b.[5] In a study conducted by Dhurat and Mathapati on microneedling shows beneficial effects on promoting new hair regrowth, even in patients who showed poor response to conventional therapy.[17]

Low-level laser therapy includes exposure of tissue to low-level infrared light at a close range. This therapy can be used as an adjuvant of MAGA therapy. A high-energy laser may disrupt cellular homeostasis, but 500–1.100 nm laser has been shown to promote tissue regeneration. Recent research assesses that laser devices show inconsistent results, providing low-level evidence. It is reported that there has been an increase in the number of hairs in some cases using this therapy.[5] In a systematic review conducted by Delaney and Zhang, a low-level laser therapy shows effectiveness in stimulating increased of hair density.[18]

Botulinum toxin injection can relax muscles, reduce pressure on blood vessels, and increase blood supply and transcutaneous pO2. The increased blood flow can also cause "cleansing" of the accumulated DHT, reducing the miniaturization signal of hair follicles. Research conducted by Singth et al. in 2018 in India discovered that botulinum toxin was an effective therapy for the management of MAGA. However, further research and trials need to be carried out.[19]

Table 1 describes several options for first-line MAGA therapy that has been approved by the Food and Drug Administration (FDA) in the form of oral finasteride 1 mg once daily and topical minoxidil 5% twice daily.[5] The Indonesian Management Guidelines of Hair Loss and Alopecia algorithm (Figure 2) have approved topical minoxidil and oral finasteride for

MAGA therapy. Patients were assessed for severity based on the Hamilton-Norwood classification, divided into mild, moderate, and severe degrees. Patients with mild androgenetic alopecia (Type I, II) are prescribed with topical minoxidil 2% or 5% for 6–12 months. Oral finasteride 1 mg or topical minoxidil 2% or 5% for patients with a moderate degree (Type IIa, III, IIIa, III vertex, IV), and hair transplantation can be considered for a severe degree of MAGA.[20]

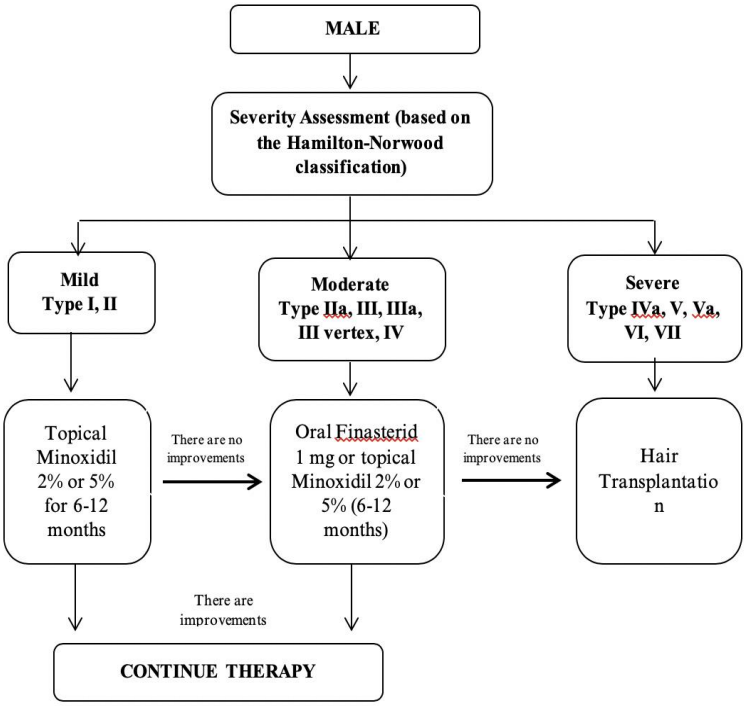

Figure 2. Male androgenetic alopecia (MAGA) Management.[20]

Androgenetic alopecia is a progressive hair loss. Therefore, the goal of therapy is to improve or even just avoid the progression of the disease. This goal can be achieved if therapy is carried out at an early stage of disease (mild to moderate). As many as 30–60% of MAGA patients show improvements after being given topical and systemic therapy, although the therapy does not completely restore the hair to its original condition. The success of therapy depends subjectively on the satisfaction of MAGA patients with the results achieved. Regardless of progression, MAGA can cause depression and affect the patient's psychosocial aspects.[1] It is important to understand that most of MAGA patients cannot accept their conditions well. Patients who seek help are commonly those who are under greater pressure and are not satisfied with the care that has been given.[2]

DISCUSSION

Male androgenetic alopecia (MAGA) is the most common hair loss in males. This disorder is influenced by several factors, such as genetic susceptibility, hormonal, hair cycle dynamics,hair follicle miniaturization, inflammation, and destruction of the arrector pili muscle. Currently, the MAGA-approved therapeutic modalities of the FDA are topical minoxidil and oral finasteride. Hair loss in MAGA is progressive, so the goal of therapy is to improve the clinical presentation and prevent disease progression. This can be achieved if the therapy is carried out at an early stage of MAGA (mild to moderate). Therapeutic modalities choice for MAGA is an antiandrogen, androgen-independent, and co-adjuvant therapies. Finasteride 1 mg and minoxidil 2–5 % solution are the only US FDA-approved treatment options for MAGA. Antiandrogen therapy is a drug with 5α-reductase inhibitor activities, finasteride and dutasteride. Finasteride currently being first-line drugs for MAGA. The difference between these two drugs is lies in their target. Finasteride only inhibits 5α-reductase type I, while dutasteride inhibits both types I and II.[1] A systematic review and meta-analysis study concludes that dutasteride seems to provide better efficacy in treating MAGA. Both of them show similar rates of adverse reactions, especially in sexual dysfunction.[21] Another research article said the same thing. Dutasteride was better than finasteride with similar adverse events. Therefore, dutasteride could become a treatment of choice for MAGA other than finasteride.[22]

5α-dihydrotestosterone (5α-DHT) is the most potent natural androgen. 5α-DHT elicits multiple physiological actions, including within prostate, seminal vesicles, hair follicles, skin, kidney, and lacrimal and meibomian glands. Recent emerging literature supports the role of 5α-DHT in the physiological function of the liver, pancreatic β-cell, eyes, and kidneys. Thus, inhibition of this enzyme with finasteride or dutasteride may induce a novel form of tissue-specific androgen deficiency that may contribute to a pathophysiological condition. Long-term use of this treatment may result in the development of non-alcoholic fatty liver diseases (NAFLD), insulin resistance (IR), type 2 diabetes (T2DM), dry eye disease, potential kidney dysfunction, among other metabolic dysfunctions. For these reasons, the author believes that the clinical community should recognize these new potential health risks associated with these drugs and it's time to sound the alarm.[23]

The well-known adverse event from finasteride use is sexual activity dysfunction, which is decreased libido, erectile dysfunction, and ejaculated volume, until permanent sexual dysfunction. There also mental-health-related finasteride use that is depression and suicide. Although relatively rare, gynecomastia can occur. Prostate-specific antigen (PSA) serum levels are reduced by approximately 50 % during the use of finasteride 1 mg daily because of the decreased prostatic DHT levels. For this reason, it is recommended that the PSA base levels are checked before starting treatment in men older than 50 years. However, a recent meta-analysis concluded that finasteride does not increase the risk of high-grade prostatic cancer. The benefits of finasteride in MAGA treatment require indefinite use and the possible short- and long-term adverse effects should be well-explained to patients. Dermatologists should specifically ask patients about sexual adverse effects and mood disturbances.[5]

Another treatment option is androgen-independent drugs, which are topical minoxidil, a prostaglandin analog, and ketoconazole. The combination of oral finasteride with topical minoxidil is said to increase its therapeutic effect. Minoxidil was the first and is the only topical drug approved by the FDA to treat MAGA. The exact minoxidil mechanism of action on hair growth is still unclear but is probably mediated via potassium channel opening, which leads to an increased cutaneous blood flow and enhanced levels of VEGF and hair growth promoters in the dermal papilla. The active metabolite that stimulates hair growth is minoxidil sulfate.[5] Minoxidil is considered a first-line treatment to aid hair growth, but it shows inconsistent efficacy. Some patients see a vast improvement, while others notice minimal changes. Dermatologists should educate patients that each individual may react to minoxidil differently, and hair growth occurs best with consistent medication adherence.[24] In MAGA treatment, the recommended dosage for minoxidil 5 % solution is 1 mL twice daily on dry scalp, while for minoxidil 5 % foam is half a capful twice daily. Both formulations should be left in place for at least 4 h. Patients should be treated for at least 6 months before efficacy assessment and treatment should be prolonged indefinitely to maintain efficacy. Patients should also be informed that drug interruption will cause acute hair shedding after 3–4 months.[5]

Latanoprost and Bimatoprost are synthetic prostaglandin analog of PGF2α that are originally used to decrease ocular pressure in glaucoma. PGF2α and its analog promote a transition from the telogen to anagen phase of the hair cycle. PGE2 and PGD2 have also been associated with the hair cycle. PGD2 is elevated in the scalp of balding men and inhibits hair lengthening via the GPR44 receptor. Also, it is known that PGE2 and PGF2α promote hair growth, while PGD2 inhibits this process. Prostaglandin analogs of PGF2α have been used originally to decrease ocular pressure in glaucoma with parallel effects in the growth of eyelashes, which suggests a specific effect in hair follicle activation. PGD2 receptors are located in the upper and lower outer root sheath region and the dermal papilla, suggesting that these prostaglandins play an important role in the hair cycle.[25]

Another treatment option for patients that do not adequately respond to first-line treatments is topical ketoconazole. Ketoconazole is an antifungal used topically as a 2 % shampoo to treat seborrheic dermatitis. It has anti-inflammatory properties due to its effect in decreasing *Malassezia* colonization. Ketoconazole also has antiandrogenic properties as it can interfere with steroidogenesis. Its use in combination with oral finasteride 1 mg might produce an additional decrease in scalp DHT levels.[5] A systematic review found seven articles about ketoconazole usage in alopecia, including two animal studies and five human studies. Murine studies demonstrated a significant increase in the mean ratio of hair regrowth to a denuded area in the ketoconazole treatment groups compared to controls. Human studies reported increased hair shaft diameter following ketoconazole use. One study reported a significant increase in the pilary index (percent anagen phase × diameter) following treatment. Studies also demonstrated clinical improvement of MAGA

based on photographic assessment and subjective evaluation. Topical ketoconazole is a promising adjunctive or alternative therapy in the treatment of MAGA. This systematic review suggests a randomized controlled trial to evaluate this drug.[26]

Moreover, the treatment modality is co-adjuvant therapy. The first choice is hair transplantation. This therapy is a complementary therapeutic option for patients older than 25 years with stabilized hair loss. The mechanism of action of hair transplantation is supported on the principle of donor dominance: hair follicles localized in androgen-insensitive areas keep their properties even when transplanted into the androgen-dependent scalp. This therapy is very popular, but the quality of evidence on the efficacy of hair transplantation is poor as studies have variable results due to differences in techniques and surgeon abilities as well as in the individual characteristics of the patients. In MAGA, good long-term cosmetic results can be achieved, since this technique requires preservation of hair growth over the occipital donor area, which is observed in men with AGA. It is important to highlight to the patient that the surgical treatment of AGA does not prevent the progression of the disease, reinforcing the importance of concomitant medical therapy. Topical minoxidil may speed the regrowth of transplanted follicles following the surgical procedure. Follicular unit transplantation, considered the gold standard technique, uses small follicular units, leading to a more physiological and natural result and offering a better outcome in terms of the final aspect. In the right candidate, hair transplantation can lead to a long-lasting, natural result that becomes evident 6–8 months after the procedure and the new hair will help reframe the patient's face and renew their self-confidence.[5]

Patients who do not achieve sufficient hair density with medical treatment or are not ideal candidates for hair transplantation can be offered camouflage methods. One recently developed camouflage option is micro-pigmentation. Hair extensions and wigs are other options. Camouflage methods have increasingly been accepted by patients to improve their appearance. Sometimes, a different haircut or hairstyle can be recommended which may help to give an impression of better hair density. Dermatologists should be updated about these techniques to offer patients reasonable camouflage options.[5]

Platelet-rich plasma (PRP) is an autologous preparation of plasma with a high concentration of platelets. Hair restoration has been increasing within PRP use. PRP's main components are platelet-derived growth factor, transforming growth factor, and the vascular endothelial growth factor has the potential to stimulate hard and soft tissue wound healing. In general, PRP showed a benefit on patients with androgenic alopecia, including increased hair density and quality.[27] A systematic review and meta-analysis found that PRP lacks serious adverse effects and effectively improves hair density and hair thickness in men and women with AGA.[28]

Microneedling is a minimally invasive dermatological procedure in which fine needles are rolled over the skin to puncture the stratum corneum. This therapy is used to induce collagen formation, neovascularization, and growth factor production of treated areas. It has been used in a wide range of dermatologic conditions, including AGA and alopecia areata, among others. Micro-needling has been successfully paired with other hair growth-promoting therapies, such as minoxidil, platelet-rich plasma, and topical steroids, and shown to stimulate hair follicle growth. It is thought that micro-needling facilitates penetration of such first-line medications, and this is one mechanism by which it promotes hair growth.[29]

A variety of laser and light sources have been promoted for the treatment of hair loss. The mechanism of action is not yet known. It has been hypothesized that light may activate dormant hair follicles, increase blood flow, and upregulate the production of growth factors and adenosine triphosphate that stimulate anagen hair. Low-level light therapy (LLLT) is a fairly new technique used in the treatment of AGA with different types of devices, such as a comb, hood, and helmet.[5] A systematic review from ten trials demonstrated significant improvement of the androgenic alopecia in comparison to baseline or controls when treated with LLLT. This review suggests the use of LLLT independently or as an adjuvant of minoxidil or finasteride.[30] Another systematic review concludes that LLLT appears to be a promising noninvasive treatment for MAGA and is safe for self-administration in the home setting.[16]

Nowadays, botulinum toxin injection is known to be effective for facial wrinkles.[31] As mentioned above, DHT induces TGFβ1 in dermal papilla cells to suppress follicular epithelial cell growth. Botulinum toxin type A (BTX) may inhibit TGF-β1 secretion from the dermal papilla cells.[32] A pilot study found that botulinum toxin intramuscular injection was safe and effective for the management of MAGA.[19] Another study suggests that intradermal injection of botulinum toxin could be a possible treatment option for MAGA.[32] A systematic review of six studied concludes that patient nama majalah apa satisfaction was high among all studies, but it did not demonstrate

the value of using botulinum toxin injection for MAGA. Subsequent prospective randomized controlled studies are required.[31]

MAGA is the most common progressive hair loss in males. The treatment remains a challenge for dermatologists because it is expected to achieve cosmetically significant regrowth and to slow additional hair loss. The accepted therapy currently by FDA is finasteride 1 mg and topical minoxidil 2-5%. There is a lot of treatment option other than that. This review updates other treatment options for MAGA therapy. Some of that still needs strong evidence for used widely.

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