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REVIEW

Finasteride and Its Potential for the Treatment of Female Pattern Hair Loss: Evidence to Date

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Abstract: The currently approved treatment for female pattern hair loss (FPHL) includes topical minoxidil administration; however, this treatment fails to achieve hair regrowth in some patients. Finasteride, a selective 5α-reductase inhibitor (5-ARI), may be considered as an alternative treatment. However, because of its potential teratogenic effects, clinical studies and use of finasteride for FPHL are limited. In this review, we aim to summarize the literature regarding the pharmacology, clinical efficacy, and adverse effects of oral finasteride for the treatment of FPHL and to provide novel therapeutic options including topical finasteride and dutasteride, a new generation 5-ARI, for the treatment of FPHL.

Keywords: alopecia, androgenetic alopecia, dutasteride, hair loss, topical finasteride, therapy

Introduction

Female pattern hair loss (FPHL) is a common hair condition in women characterized by diffuse hair thinning over the crown and parietal scalp with retention of the frontal hairline (Figure 1).[1,2] The prevalence of FPHL increases with advancing age, affecting 50% of women during their lifetime.[3] FPHL presents with follicular miniaturization and shortening of the anagen phase, similar to androgenetic alopecia (AGA) in men; nevertheless, the pathogenesis of FPHL remains unclear.[4] The present understanding of relationship between androgenic hormone and FPHL is controversial as evidence suggests normal hormone levels in most balding females, and there is uncertainty regarding its hereditary nature.[5]

Various treatment options have been attempted to treat FPHL. The only agent approved by the US Food and Drug Administration (FDA) is topical minoxidil.[6,7] Other treatment options currently available include low-level laser therapy, fractional laser therapy, platelet-rich plasma, human follicle stem cells and hair transplantation.[8–12] Nevertheless, the treatment outcome may not be satisfactory in some patients. Finasteride, an inhibitor of type II 5α-reductase enzyme, is currently indicated for AGA in men. It has been increasingly used as an off-label treatment for FPHL.[13] Despite its potential teratogenic effect, several publications on finasteride in FPHL have shown positive results. Therefore, this review aims to summarize the pharmacology, therapeutic efficacy as well as safety of oral finasteride for the treatment of FPHL. Furthermore, we provide novel therapeutic options of 5α-reductase inhibitor (5-ARI), namely topical finasteride and oral dutasteride.

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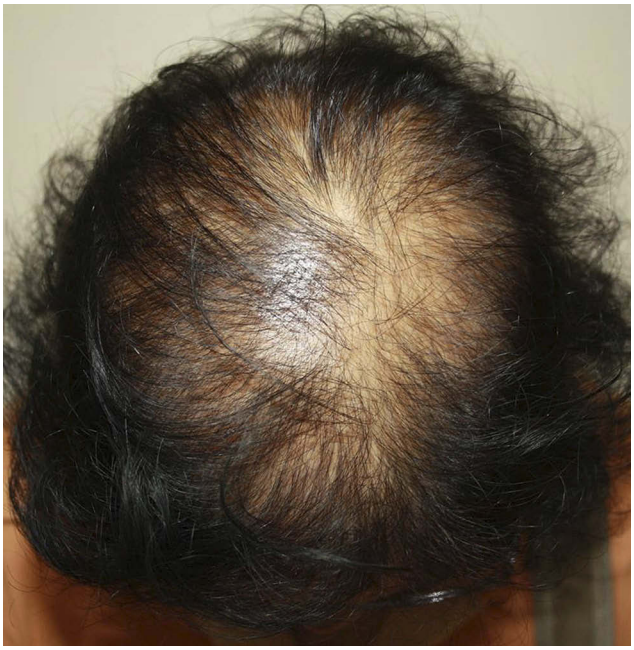

Figure 1 Female pattern hair loss: hair thinning mostly confined to the crown with retention of frontal hairline.

Pharmacology of Finasteride

Finasteride is a synthetic 4-azasteroid compound ($C_{23}H_{36}N_2O_2$) (Figure 2) that competitively inhibits type II 5α-reductase, resulting in the prevention of the conversion of testosterone to dihydrotestosterone (DHT) in the skin, liver and prostate gland.[14] A study showed that oral finasteride reaches its maximum plasma concentration approximately 1–2 hrs after ingestion, while achieving the steady-state within three days. Finasteride suppresses scalp DHT levels by 43% at 28 days, up to 65% at 42 days of treatment with finasteride 5 mg daily in patients with AGA.[15] To our knowledge, no study evaluating the changes of scalp DHT in women with FPHL has been reported. The bioavailability of finasteride is 80% and it is not meaningfully influenced by food.[16] Finasteride can cross the blood-brain barrier; however, it only insignificantly distributes to cerebrospinal fluid.[17] Finasteride has no effect on luteinizing hormone (LH) and follicle-stimulating hormone (FSH), while it slightly increases circulating testosterone levels but remains within the normal physiologic range.[18]

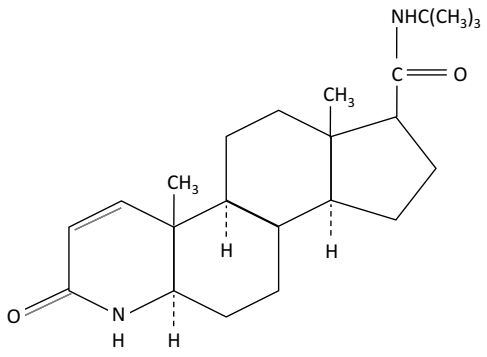

Figure 2 Chemical structure of finasteride.

Finasteride is exclusively metabolized via cytochrome P450 3A4 in the liver.[19] The major metabolites (ω-hydroxyfinasteride and a monocarboxylic acid derivative) contain less than 20% of 5α-reductase inhibitory effect and are eliminated through bile.[17] After 7 days of administration, approximately 57% of the dose is excreted in the feces, and approximately 39% is excreted in the urine.[17] DHT returns to pretreatment levels by approximately 14 days after cessation.[20] Despite a lack of studies in patients with abnormal liver functions, it is recommended that finasteride be used with caution in patients with hepatic impairment as it is preferentially metabolized in the liver. In healthy young adults, the mean half-life of finasteride in plasma is 6 hrs. The mean elimination half-life in elderly subjects (age ≥70 years) is approximately 8 hrs, not significantly different from that of subjects who are 45–60 years of age.[21] Therefore, no dosage adjustment is required for the elderly. Although finasteride is primarily metabolized by hepatic enzymes, it does not appear to interfere with the enzyme system. Interaction studies showed that there are no drug interactions between finasteride and antipyrine, digoxin, propranolol, theophylline, and warfarin. Moreover, there is no evidence of clinically significant interactions with concomitant use of drugs such as anti-convulsants and nonsteroidal anti–inflammatory drugs.[17]

Finasteride is classified as pregnancy category X, ie, it is contradicted in women who are or may become pregnant.[17] An animal study showed that finasteride led to dose-dependent development of hypospadias in male offspring, and that the abnormal development of external genitalia is an expected aftereffect from inhibition of type II 5α-reductase similar to male children with genetic 5α-reductase deficiency.[22] No developmental abnormalities were seen in female fetuses. Finasteride is also prohibited in lactating women because of its potential risks in male infants, despite the unavailability of data on its excretion in human milk. Finasteride can interfere with the estrogen/testosterone balance, leading to potential risk of estrogen-mediated malignant transformation; it should be avoided in those who have family history of breast cancer.[23]

Therapeutic Efficacy of Finasteride in Female Pattern Hair Loss

Finasteride has been commonly used in men with AGA and benign prostatic hyperplasia, and is progressively prescribed in women with hyperandrogenism-associated conditions including acne vulgaris, hirsutism and pattern alopecia induced by testosterone in female-to-male transgender patients.[13,24] Nevertheless, studies of the efficacy of oral finasteride in FPHL are limited because of the potential risk of teratogenicity in the male fetus.[5] The dosage of finasteride for FPHL ranges from 1 to 5 mg daily. Case reports and series using various regimens showed increased hair density and reduction of hair shedding in pre- and postmenopausal women with or without hyperandrogenism.[25–29] Evidence from clinical trials of finasteride in FPHL is summarized in Table 1.

A multicenter, double-blind, randomized controlled trial (RCT) of 137 postmenopausal women with mild-to-moderate FPHL reported no significant differences in changes of hair loss by hair count between finasteride 1 mg and placebo after 1 year of treatment.[3] Patient and investigator assessments, as well as global photographic assessment (GPA), failed to show any significant improvement from baseline with respect to hair growth, hair appearance or stabilization of hair loss in both groups. Histological examination of scalp biopsy specimens correspondingly showed no significant differences between the groups with respect to alterations from baseline regarding terminal-to-vellus hair ratio and anagen-to-telogen ratio.[30,31]

A subsequent open-label, uncontrolled study using low-dose finasteride (1.25 mg) in 18 normoandrogenic FPHL patients similarly failed to demonstrate increasing hair density evaluated by phototrichogram.[32] No clinical response was observed by patient self-assessment and physician's GPA compared to baseline. In contrast to these failures, a retrospective cohort demonstrated the efficacy of finasteride 1.25 mg or dutasteride 0.15 mg daily in 120 FPHL patients.[33] Each group contained 60 patients, half of whom were aged 50 years or above. After 3 years of treatment, 82% of patients in the finasteride group and 83% of patients in dutasteride group demonstrated improvement in hair thickness compared to the baseline. GPA by physicians at the vertex and center of the scalp reported increased hair density and scalp coverage by 67% in the finasteride group and 66% in the dutasteride group. Nevertheless, the study did not objectively measure hair density or compare the effectiveness with placebo.

Medium-dose finasteride (2.5 mg daily) also proved to be efficacious in normoandrogenic postmenopausal women with FPHL. Trueb et al conducted a small prospective study using finasteride 2.5 mg daily in four women and 5 mg daily in one woman who had been previously treated with topical minoxidil alone or combined with cyproterone acetate (CPA), topical estradiol, and systemic estrogens without clinical response.[34] By 6 months, all patients reported the stabilization of hair loss and four patients self-reported noticeable hair growth. These findings were confirmed by investigator assessments and photography evaluations. A single-center, uncontrolled, prospective trial further evaluated the efficacy of the medium-dose finasteride in 37 pre-menopausal women with FPHL in the absence of hyperandrogenism.[30] Finasteride was given for 12 months at a dose of 2.5 mg daily with concomitant use of an oral contraceptive containing drospirenone and ethinyl estradiol to prevent pregnancy. After 12 months, global photography displayed the enhancement of hair density in 62% of patients. Hair density scores assessed by computerized videodermoscopy significantly increased compared to baseline in 32% of patients. On self-administered questionnaire, 78% of the patients reported improvement, while 22% reported stabilization of hair loss. Nonetheless, the clinical outcome of 2.5 mg finasteride was inconclusive as improvement in hair density may have resulted from the antiandrogenic effect of the oral contraceptive.

High-dose finasteride (5 mg daily) has been investigated for efficacy in treatment of FPHL. A retrospective study using self-filled questionnaires evaluated effectiveness of finasteride 5 mg daily for unknown duration in 12 patients with FPHL or acne vulgaris.[35] Five of six patients with FPHL reported to benefit from the treatment. Carmina et al conducted a subsequent single-center, open-label, RCT in hyperandrogenic pre-menopausal women with FPHL.[36] Forty-eight patients were randomly assigned into four groups which consisted of finasteride 5 mg daily, CPA 50 mg (day 5–15 of menstrual cycle) with ethinyl estradiol 25 μg/day (day 5–25 of menstrual cycle), flutamide 250 mg daily, and no treatment for 1 year. All patients including controls showed decreases in Ludwig scores at the end of treatment. Flutamide was the only agent that gave a statistically significant reduction of Ludwig scores and provided the greatest clinical response in terms of slowing hair loss as assessed by the patients. Using a 7-point scale, flutamide demonstrated the best, although not significant, improved hair growth compared

Table 1 Clinical Studies of Finasteride in the Treatment of Female Pattern Hair Loss

Author, Year	Study Design	N	Finasteride Regimen	Duration of Treatment	Population	Results	Side Effects
Oral finasteride							
Price et al, 2000[3]	RCT	137	1 mg/day vs placebo	12 months	41–60 years; normoandrogenic post-menopausal women, Ludwig scores III–IV	• Decreased mean hair counts in both groups (−8.7 hairs in finasteride group vs −6.6 hairs in placebo group) • No significant changes of hair loss by patients' & physicians' GPA, & histologic examination • Decreased serum DHT & androstanediol glucuronide	Folliculitis (n=1)
Carmina et al, 2003[36]	RCT	48	5 mg/day vs CPA with EE vs flutamide 250 mg/day vs none	12 months	25±2 years; hyperandrogenic pre-menopausal women, Ludwig scores I–III	• Flutamide significantly decreased Ludwig scores • No significant changes in Ludwig scores and clinical evaluation in finasteride group	• None in finasteride group • Elevated liver enzyme in flutamide group (n=2)
Trueb et al, 2004[34]	Uncontrolled, prospective	5	2.5 or 5 mg/day	18 months	55–69 years; normoandrogenic post-menopausal women, Ludwig score I, Hamilton-Norwood VII, Olsen type	• Stabilization of hair loss (n=5) • Noticeable hair growth (n=4)	None
Iorrizo et al, 2006[30]	Uncontrolled, prospective	37	2.5 mg/day + OCP	12 months	19–50 years; normoandrogenic pre-menopausal women, Ludwig scores I–II, Olsen type	• 62% of patients improved GPA • Increased mean hair density from 4.5 to 4.8	None
Kohler et al, 2007[35]	Retrospective survey	12	5 mg/day	Unknown duration	26–76 years; women with unspecified status	• 93.3% of patients reported improvement	None
Yeon et al, 2010[37]	Uncontrolled, prospective	87	5 mg/day	12 months	44.4±10.6 years; normoandrogenic pre- & post-menopausal women, Ludwig scores I–III	• 18.9% increased hair density • 9.4% increased hair diameter • 81.4% of patients improved GPA	Headache, irregular menstruation, dizziness, increased body hair (n=4)
Kim et al, 2012[32]	Uncontrolled, prospective	18	1.25 mg/day	7 months	46.3±12.8 years; normoandrogenic pre- & post-menopausal women	• 5.87% increment of hair density • 11.8% increment of hair diameter • 14.2% of patients improved according to patient's and physician's GPA	NA

Oliveira-Soares et al, 2013[38]	Uncontrolled prospective	40	5 mg/day	18 months	<60 years; normoandrogenic post-menopausal women, Ludwig scores I–III	• Approximately 50% of patients had major improvement from patient's GPA at 6, 12 & 18 months • Approximately 40% of patients had major improvement assessed by physicians at 6, 12 & 18 months	Maintained loss of libido (n=4), elevated liver enzyme (n=1)
Boersma et al, 2014[33]	Retrospective	120	1.25 mg/day or dutasteride 0.15 mg/day	3 years	16–84 years; pre- & post-menopausal women, Ludwig scores I–II	• Improved hair thickness in 81.7% of patients in finasteride group, 83.3% patients in dutasteride group • Improved hair density from GPA in 68.9% of patients in finasteride group vs 65.6% of patients in dutasteride group	NA
Topical finasteride							
Mazzarella et al, 1997[43]	Pilot RCT	52	0.005% topical finasteride twice daily	16 months	18–38 years; men & pre-menopausal women, Hamilton-Norwood scales I–III in men, Ludwig scores I–II in women	• Progressive decreased hair counts from wash test from 49.8 ± 5.9 to 45.2±7.5 at 6 months and 36.8±8.1 at 16 months • Clinically improved hair density and increased hair regrowth	None
Suchonwanit et al, 2019[44]	RCT	30	0.25% topical finasteride + 3% MNX vs 3% MNX twice daily	6 months	56.8±6.6 years (finasteride+MNX) vs 59.8±7.7 years (MNX); post-menopausal women, Ludwig scores I–III	• Mean increased hair diameter of 11.9 μm in finasteride+MNX group & 7 μm in MNX group at 6 months • Mean increased hair density of 24.7 hairs/cm^2 in finasteride+MNX group & 21.9 hairs/cm^2 in MNX group at 6 months • Approximately 93% of patients had improved GPA in both groups • Decreased serum DHT level in finasteride+MNX group	Folliculitis

Abbreviations: CPA, cyproterone acetate; DHT, dihydrotestorone; EE, ethinyl estradiol; GPA, global photographic assessment; OCP, oral contraceptive; MNX, minoxidil; NA, not available; RCT, randomized controlled trial.

to CPA, finasteride, and controls. Two patients had elevated liver transaminase from flutamide, while no adverse effects were noted in the finasteride and CPA groups. The authors concluded that finasteride was ineffective for treatment of FPHL.

By contrast, a non-randomized, uncontrolled study of high-dose finasteride in 87 normoandrogenic, pre- and post-menopausal women with FPHL reported clinical improvement.[37] Eighty-six patients completed 12 months of treatment with finasteride 5 mg daily, whereas one patient discontinued because of headache. At the end of the study, phototrichograms demonstrated significantly increased hair density by 18.9% and hair diameter by 9.4% compared to baseline. GPA showed clinical improvement in 81% of patients. There was no significant difference in the clinical response between pre- and post-menopausal groups. The authors concluded that finasteride might be effective and safe for FPHL in women without hyperandrogenism. Another uncontrolled prospective study on efficacy of finasteride 5 mg daily was conducted in 40 normoandrogenic post-menopausal FPHL patients. All patients were divided into three age groups: <60 (n = 22), 60–70 (n = 13), and >70 years (n = 5).[38] Fifty-five percent of patients reported having major improvement, and 30% reported having moderate improvement with as little as 6 months of treatment. Although the improvement tendency was observed over time, it was relatively constant over 12 and 18 month-periods. Patients who were >70 years of age demonstrated poorer clinical responses than did the other two younger groups. According to pre-existing data, high-dose finasteride appears to be more efficacious than the lower doses for the treatment of FPHL.

Mechanism of Finasteride in Female Pattern Hair Loss

Because FPHL is a complex disease, the mechanism by which finasteride improves hair loss is not well established. A study in mice proposed that DHT may downgrade insulin-like growth factor-1 (IGF-1) expression by inhibiting the release of calcitonin gene-related peptide that interacts with the androgen receptors, thereby preventing hair growth.[39] These findings suggested that finasteride might be associated with increasing IGF-1 production in the dermal papillae by decreasing DHT levels.[40] Rushton et al found that finasteride administration resulted in increased mean hair density without insignificant changes of vellus hair counts.[41] The observed hair regrowth in FPHL may be supported by reactivating telogen/kenogen follicles into anagen follicles rather than vellus-to-terminal hair transformation. Further studies are needed to determine the mechanism of finasteride in FPHL. Concerning contradictory efficacy in pre-existing literatures, oral finasteride may be served as an alternative modality in FPHL patients who fail minoxidil treatment. We suggest using finasteride in postmenopausal women to avoid potential teratogenic effects.

New Formulation of Finasteride

A topical formulation has been proposed as a new treatment modality to minimize the unwanted systemic side effects of oral finasteride, particularly in child-bearing aged women. The ideal formulation of topical finasteride is high skin penetration and low systemic absorption. Topical finasteride has recently shown to be effective in males with AGA.[42] Nevertheless, studies on efficacy of topical finasteride in females have been limited. Mozzarella et al conducted a placebo-controlled trial with 0.005% finasteride solution twice daily for 16 months in 52 patients, including 28 men and 24 pre-menopausal women.[43] The finasteride-treated group showed a significantly greater reduction of hair shedding from the 6th month of treatment and progressively continued throughout the 16-month period. At the end of study, some finasteride-treated scalps demonstrated gradual increases in hair density.

A recent single-center, double-blind RCT compared the efficacy between topical 3% minoxidil solution alone and in combination with 0.25% finasteride for treatment of 30 post-menopausal FPHL patients.[44] The combined treatment group showed significantly superior results compared to the minoxidil monotherapy group in terms of increased hair diameter at weeks 24, while increased hair density was progressively observed over time from weeks 8 in both groups with similar time-courses. At week 24, clinical improvements as assessed by GPA from blinded dermatologists and participants were reported in approximately 93% of participants in the combined treatment group but were not significantly different from the minoxidil group. The authors agreed with the previous literature that adding 0.25% finasteride to minoxidil solution showed evidence of systemic absorption by lowering serum DHT level after 24 weeks of treatment. Nevertheless, DHT levels remained in the normal range throughout the study.

Topical application with finasteride is a promising treatment for FPHL with favorable results. Many aspects of topical finasteride are required for further investigations,

including delivery systems, concentrations, and regimens. Because several studies used various formulations, a comparative study between each formulation (eg, gel versus solution) should be performed to determine the best use of topical finasteride.

Tolerability of Finasteride

Side effects of finasteride have been relatively well documented in male AGA patients. These side effects include ejaculation disorders, decreased libido, erectile dysfunction, breast tenderness and enlargement, dizziness, allergic reactions, increased liver enzymes, and depression.[45,46] Post-finasteride syndrome, characterized by persistent sexual, neurological, and physical adverse reactions, has also been described.[45,47] Because finasteride is not widely used in the treatment of FPHL, the side effects in women are not well elucidated.

Oral finasteride given at low-to-medium dose did not demonstrate any complications. Oral finasteride 1 mg daily reduced serum levels of DHT and androstanediol glucuronide in postmenopausal women without clinical change, while had no effect on other hormones including serum testosterone, LH, FSH, thyroid-stimulating hormone and prolactin. High-dose oral finasteride disturbed women with side effects analogous to those of men. In five patients, Kohler et al found one patient with decreased libido, one with dry skin and one with mild acne.[35] In a cohort study of 87 patients, headache, irregular menstruation, dizziness and increased body hair growth were reported in four patients, but were minimal and transient.[37] Another cohort study by Oliveira-Soares et al noted persistent libido reduction in four patients and increased liver enzyme levels in one patient.[38]

By contrast, no systemic side effects have been reported with the use of topical finasteride in FPHL patients except suppression of serum DHT levels. Minimal local side effects such as pruritus and irritation have been reported, but with an excellent tolerability.[43,44] Because of limited data, further clinical trials with long-term use should be performed to confirm the safety of topical finasteride in women.

New Generation of 5α-Reductase Inhibitors

Dutasteride is a second-generation 5-ARI that inhibits both type I and II 5α-reductase isoenzymes. It is three times more potent at inhibiting type I enzyme and 100 times more potent at inhibiting type II enzyme than is finasteride.[48] Although dutasteride is not currently FDA-approved for pattern hair loss, recent studies have pointed to its ability to serve as a therapeutic agent.[49,50] A study in 416 men with AGA found that dutasteride 2.5 mg daily had efficacy superior to that of finasteride 5 mg daily and placebo in terms of increasing hair counts and scalp appearance by GPA after 12 and 24 weeks of treatment.[49] By contrast, pre-existing studies of dutasteride for treatment of FPHL are limited. A case study of a 46-year-old woman with FPHL who had little response to minoxidil and finasteride reported clinical improvement after 6 months of dutasteride 0.5 mg daily and marked improvement after 9 months of therapy.[51] Boersma et al found that dutasteride 0.15 mg daily was more effective than finasteride 1.25 mg daily as it caused higher mean hair thickness at the vertex and the center of the scalp in patients younger than 50 years.[33] These available data support the efficacy of dutasteride for the treatment of FPHL; nevertheless, further studies comparing dutasteride with finasteride and placebo are necessary to determine its real safety and efficacy.

Conclusion

This review summarizes the pharmacology and presents potential uses of finasteride in women, especially in the postmenopausal-aged group, and those wishing to avoid systemic side effects. Dutasteride, a new generation of 5-ARIs is also discussed. The studies we reviewed suggest that finasteride could be considered as an alternative treatment option for FPHL. Nevertheless, many questions regarding finasteride, particularly with respect to topical formulations, are yet to be answered. Further studies are encouraged to determine the maximum therapeutic efficacy and to evaluate the consequences of finasteride use in FPHL.

Statement of Ethics

The patient provided written informed consent to perform all necessary investigations, to take clinical photographs, and to use them for research purposes and publication.

Disclosure

The authors report no conflicts of interest in this work.

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