Management of female-pattern hair loss

REBECCA MANN

Female-pattern hair loss is a common and distressing condition that can lead to a progressive and irreversible decline in scalp hair density if left untreated. This article discusses the causes, characteristics and management of hair loss in women.

Female-pattern hair loss is a common and distinctive form of non-scarring hair loss that affects adult women. By the age of 50 years, around 40 per cent of women will show signs of hair loss, while less than 45 per cent of women will reach the age of 80 years with a full scalp of hair. If untreated, female-pattern hair loss can lead to a progressive and irreversible decline in the density of the scalp hair. Hair is a key part of our identity and there is a social stigma attached to female baldness. Female-pattern hair loss can be a distressing condition that can cause significant anxiety and emotional turmoil. The GP has an important role in diagnosing hair loss and directing the patient to the appropriate treatment and resources.

The healthy adult scalp contains about 100,000 hairs, of which 90 per cent are actively growing. They are known as ‘anagen’ hairs and they cannot be easily pulled out as the hair is anchored deeply into the subcutaneous fat of the scalp. Hair constantly regenerates on the scalp; each hair shaft may persist on the scalp for between three and seven years, before shedding and being replaced by a new hair shaft. The anagen phase lasts for most of this three to seven-year period and is followed by a ‘catagen’ phase of approximately two weeks. This is the apoptosis (programmed cell death) of the hair shaft. The three-month resting or ‘telogen’ phase then follows. Telogen hair can be pulled out relatively easily, and a normal scalp will lose around 100 telogen hairs per day.

Beside the ratio of anagen to telogen hair on the scalp, the diameter of the hair follicles also plays a role in scalp coverage. Vellus hairs have a diameter of less than 0.03mm, compared to greater than 0.06mm for terminal hairs. Optimal scalp hair growth and coverage of the scalp are provided by the anagen and terminal hairs.

Causes

Female-pattern hair loss has a strong genetic predisposition, with a polygenic inheritance from either or both parents. It has long been thought that female-pattern hair loss was the counterpart to male-pattern hair loss, and was originally...
Female-pattern hair loss

Male-pattern hair loss occurs as a consequence of the effects of dihydrotestosterone (a potent metabolite of testosterone) on susceptible hair follicles. Dihydrotestosterone binds to androgen receptors in hair follicles, resulting in the upregulation of genes responsible for the gradual transformation of terminal hair follicles to miniaturised hair follicles. It was initially thought that a similar pathophysiology was responsible for female-pattern hair loss, as women with hyperandrogenic disorders such as polycystic ovary syndrome had features of early-onset female-pattern hair loss. However, the majority of women diagnosed with female-pattern hair loss have normal androgen levels, thus indicating that the cause is not solely related to androgen hormones.

Several studies have indicated links between female-pattern hair loss and both body mass index and type 2 diabetes mellitus, which are associated with insulin resistance. Earlier puberty, fewer childbirths and use of the oral contraceptive pill are also found to be positively associated with developing female-pattern hair loss. Conversely, prolactin (which is elevated during breastfeeding) might be associated with decreased risk of the condition, although this has not been conclusively proven. The role of oestrogen is uncertain; female-pattern hair loss is more common after the menopause, suggesting that oestrogen has a stimulatory role in hair growth.

**Signs and symptoms**

Insidious, diffuse thinning of the hair on the scalp due to increased shedding or a reduction in hair volume is the most common presenting symptom (see Table 1). When women with noticeable signs and symptoms present to the GP up to around 50 per cent of hair loss may have already occurred. There is a progressive reduction in the density of the terminal hairs, with varying distributions of hair loss. The frontal scalp and vertex of the scalp are the primary sites of involvement. The frontal hairline is usually preserved.

The exact pattern of the hair loss may vary. Frontal scalp thinning produces a ‘Christmas tree-type’ pattern where the hair is parted at the midline. Diffuse central thinning may be the prominent feature in some women, and a large proportion of women may exhibit bitemporal thinning. The occipital scalp is usually spared and complete loss of all scalp hair is rare. The hair loss is not constant and women may notice accelerated phases lasting three to six months, followed by quiescence lasting six to 18 months.

Scarring, itching, soreness, scaling and inflammation of the scalp are not features of female-pattern hair loss.

**Susceptibility**

Advancing age has a strong correlation with the development of female-pattern hair loss. There are two peaks to the onset of hair loss: in the third and fifth decades. Early-onset female-pattern hair loss is linked to significant hair loss, especially compared with women who present with postmenopausal hair loss. Women with a family history of male-pattern hair loss are also more susceptible.

There are a number of conditions that may be associated with female-pattern hair loss, including polycystic ovary syndrome, ovarian and adrenal tumours. A history of the six months preceding the hair loss should be explored with the patient, as significant physical or emotional stress can affect the hair follicles. This may suggest a reversible cause for the hair loss, known as telogen effluvium. This condition does not require any treatment and hair growth should return to normal within a few months.

**Management**

Treatment is available for female-pattern hair loss (see Table 2), though there is no cure. Patient expectations should be managed accordingly. The aim of treatment is to slow the rate of progression of hair loss, rather than promote hair regrowth. However, self-confidence may be improved by trying a different hairstyle or hair products that thicken the hair.

### First-line therapy

- Minoxidil 2% cutaneous solution twice daily – apply to affected scalp indefinitely
- Minoxidil 5% cutaneous foam once/twice daily – apply to affected scalp indefinitely (dermatologist referral recommended)

### Second-line therapy

- Spironolactone 100mg twice daily for at least six months (dermatologist referral recommended)

**Table 2. Treatment of female-pattern hair loss**

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**Table 1. Key features of female-pattern hair loss and examination findings**

<table>
<thead>
<tr>
<th>Key features</th>
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<tbody>
<tr>
<td>Increased hair shedding</td>
</tr>
<tr>
<td>Reduced hair volume</td>
</tr>
<tr>
<td>Frontal scalp involved</td>
</tr>
<tr>
<td>Vertex of scalp involved</td>
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<tr>
<td>Bitemporal thinning</td>
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</table>

<table>
<thead>
<tr>
<th>Examination findings</th>
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<tr>
<td>Terminal hair loss – usually in the susceptible areas</td>
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<tr>
<td>Miniaturised hairs (shorter and thinner hairs)</td>
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<tr>
<td>Preservation of the frontal hairline</td>
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<tr>
<td>No scale, flaking or inflammation present</td>
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referred to as female androgenic alopecia in the literature. However, the role of androgens is less proven in female-pattern hair loss compared with male-pattern hair loss.

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strengths, in pharmacies or on private prescription. A 5% cutaneous foam preparation is now available for women and most dermatologists agree that this preparation gives greater sustained hair growth. It must be continued for at least 12 months. Women may not notice the effect until at least eight weeks into the course of treatment.

Minoxidil is a potassium-channel opener and vasodilator, but the mechanism by which it improves female-pattern hair loss is not completely understood. One theory is that the drug lengthens the anagen phase of the hair follicles, shortens the resting phase and induces enlargement of the miniaturised follicles, and therefore the conversion of these hairs to terminal hairs.

Potential local adverse effects of minoxidil include scalp pruritus, flaking and facial hypertrichosis. The low risk of scalp irritation with use of the 5% cutaneous foam formulation may be related to the absence of propylene glycol, a common irritant and a potential contact allergen.

Second-line treatment
Antiandrogen therapy is recommended as a second-line treatment. Systemic medications that inhibit androgen production or its effects, such as spironolactone, cyproterone acetate and flutamide are available off licence if women respond poorly to monotherapy with minoxidil or have female-pattern hair loss related to hyperandrogenism.

Spironolactone is generally recommended as the first choice in second-line treatment. It is an aldosterone antagonist that competitively blocks androgen receptors and weakly inhibits androgen synthesis. There is a reasonably large body of data to support the efficacy of spironolactone and, similar to minoxidil, it must be continued for at least six to 12 months to assess the effect on the hair. Flutamide and cyproterone acetate are infrequently used in the UK as there is a paucity of data available and the dosing regimens are not standardised.

Finasteride is not recommended in women as there is currently little evidence to support its efficacy.

Role of the GP
Women may present to the GP with varying stages of hair loss, that may or may not be accompanied by psychological distress. Hair loss can be difficult for women to deal with, especially younger women, and GPs should signpost patients to counselling services if necessary or to online resources, such as Alopecia UK or NHS Choices, for further information about female-pattern hair loss.

- Is there a family history of hair loss?
- Are there any scalp symptoms or signs apart from hair loss?
- Are there any symptoms of hyperandrogenism or virilisation?
- What is the rate of progression of hair loss?
- Have there been significant health or life changes in the last six months?

Table 3. Key questions to ask patients

Clinical examination of the scalp can provide all the necessary information to diagnose female-pattern hair loss. Use of a dermatoscope will demonstrate the varying diameters of the hair shafts, which can then be compared to the normal follicular pattern. Key questions to ask patients are outlined in Table 3.

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Other causes of hair loss should be excluded, including telogen effluvium, particularly if the hair loss is very rapid. Systemic lupus erythematosus can occasionally present in this way, as can hypothyroidism, and therefore free thyroxine (T4) levels should be measured. Stored ferritin levels should also be checked, to rule out ferritin deficiency and iron-deficiency anaemia presenting with hair loss is a separate diagnosis to female-pattern hair loss. There is little evidence to support iron deficiency accelerating female-pattern hair loss, although iron supplementation should be given in those women with female-pattern hair loss who have an incidental finding of low ferritin. Before confirming the diagnosis, the GP should ask about the woman’s menstrual cycle, particularly the duration and the frequency of her periods, as the hair loss may be linked to changing oestrogen levels.

Dermatological opinion is recommended before antiandrogen treatment is commenced. Once treatment has commenced, women who begin a course of spironolactone should have their serum potassium levels checked around six weeks into the dosing regimen, as hyperkalaemia is a possible side-effect. Baseline blood pressure should be checked and then routinely every six months along with a serum potassium level. Women should be counselled on the need for effective contraception while taking antiandrogenic medication as feminisation of a male foetus can occur if the woman conceives during a course of antiandrogenic medication.

Conclusion
Female pattern hair loss, while relatively common, can be a difficult condition to cope with. As such, the GP has a central role in supporting the patient. Women of all ages may visit the GP with varying severity of hair loss, most of whom will not have deranged androgen profiles. General practitioners should feel confident in diagnosing female pattern hair loss and initiating

KEY POINTS
- Female-pattern hair loss is a common nonscarring form of hair loss
- Females of all ages can be affected
- It presents with a slow, progressive transition of terminal hairs on the frontal scalp and/or vertex of the scalp to shorter, thinner hairs and vellus hairs. The process results in a visible reduction in hair coverage on the scalp
- The majority of patients with female-pattern hair loss do not have abnormal androgen profiles
- Female-pattern hair loss is a clinical diagnosis that can be made in primary care
- GPs can diagnose and treat uncomplicated cases without dermatological referral
first-line treatment, as any further delay in care may worsen the hair loss and increase the emotional distress for the patient.

References

Declaration of interests
None to declare.

Dr Mann is a specialty doctor in oral and maxillofacial surgery at Hampshire Hospitals NHS Foundation Trust

POEMs

Blood pressure lowering and statins not synergistic for cardiovascular risk reduction

Clinical question:
In persons at intermediate risk for a cardiovascular (CV) event, does medication to reduce blood pressure and cholesterol reduce the likelihood of CV events?

Bottom line:
This large and well-executed trial confirms that treating elevated blood pressure reduces the likelihood of CV events and that statins provide a consistent relative reduction in risk, regardless of the baseline risk. But the absolute risk reduction is lower and the number needed to treat (NNT) is higher when the person starts at a lower baseline risk. Statins at a dose similar to that given in this study reduce the likelihood of a CV event by approximately 25 per cent. So, if you are starting at a 10-year risk of 20 per cent, that gets you down to 15 per cent, for an NNT of 20. Pretty good. If your 10-year risk is 10 per cent, as in this population, it reduces your likelihood to 7.5 per cent, for an NNT of 40. If your 10-year risk is only 5 per cent, it takes you down to 3.75 per cent, for an NNT of 80. These numbers should be the basis of our discussions with patients about statins. *(LOE = 1b)*

Reference:


Synopsis:
This is part of a large clinical trial that evaluated the simultaneous effect of blood pressure lowering and cholesterol lowering in patients at intermediate risk for CV disease. The researchers enrolled men older than 55 years and women older than 65 years with at least one CV risk factor, and women older than 60 years with two risk factors. The patients had an average 10-year risk of having a CV event of 10 per cent, which the authors categorised as intermediate risk. Patients were randomised into one of four groups: candesartan-hydrochlorothiazide (HCTZ) plus rosuvastatin, candesartan-HCTZ plus placebo, rosuvastatin plus placebo, or placebo plus placebo.

The primary outcome was a composite of CV death, nonfatal myocardial infarction, and nonfatal stroke; patients who received candesartan-HCTZ plus rosuvastatin had a somewhat lower likelihood of this outcome, but the likelihood was similar to that in the rosuvastatin study *(3.6 vs 5.0 per cent; p=0.005; NNT = 67 over 5.6 years).* There was no change in CV deaths or all-cause mortality.