

lymphatics. Surgery should be considered in low-risk patients with significant symptoms such as malnutrition.³

We did not arrange lymphoscintigraphy for the patient because his parents did not give consent. We are managing the patient with conservative treatments. Further close follow up of the clinical condition is necessary.

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A woman with iatrogenic androgenetic alopecia responding to finasteride

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SIR, Finasteride, a type II 5 α -reductase inhibitor, has long been proposed to treat androgenetic alopecia in women.¹ Although female hair loss associated with hyperandrogenism can respond to finasteride,² there has been no report on its efficacy for rare cases of androgenetic alopecia that develops in women undergoing androgen therapy. We describe a woman receiving exogenous androgen supplementation who developed Hamilton type hair loss and responded well to a medium-high dose of finasteride (2.5 mg daily).

In 2005, we began treating a 47-year-old woman with a 2-year history of progressive scalp hair loss. At the age of 24 years, she was diagnosed with a right ovarian dysgerminoma presenting with right lower abdominal distension and underwent a total hysterectomy and bilateral salpingoophorectomy followed by two courses of chemotherapy [Adriamycin[®] (doxorubicin) and cisplatin]. She was then given Disemone[®] (4 mg estradiol valerate, 90.2 mg testosterone enanthate) monthly for her surgically menopausal status, which caused discomfort including hot flushing. In 2003, her hormonal supplementation was increased to nearly twice monthly, better

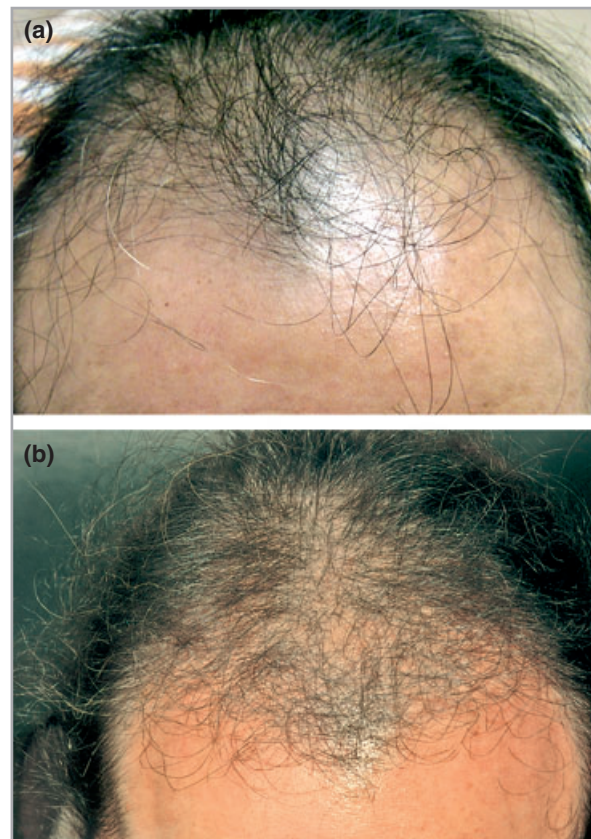


Fig 1. Clinical pictures of the patient before treatment (a) and after 10 months of therapy with finasteride (b).

to control her menopausal symptoms. Afterwards, progressive hair thinning on the scalp developed.

In 2005, she presented to our dermatology clinic for evaluation of progressive hair loss. Physical examination revealed a nonscarring Hamilton type IV–V alopecia on the vertex with significant frontotemporal recession (Fig. 1a). The hair shafts and scalp skin were normal. She also had clinical signs of hyperandrogenism, including facial hirsutism, muscular body habitus, and a deepened voice. Thus, we suspected that she had developed an iatrogenic androgen-induced alopecia.

Reviewing the literature, we found no evidence that finasteride might have an adverse effect on a patient with dysgerminoma who had undergone complete surgery and had been recurrence-free for 23 years. She was treated with a medium-high dose of finasteride (2.5 mg daily). After the first 6 months, her hair loss stabilized. By 10 months, despite still receiving androgen supplementation, she had noticeable improvement of hair coverage on the scalp (Fig. 1b, Hamilton type III) as compared with that at baseline (Fig. 1a). She also reported decreased hirsutism and a higher pitch of her voice.

Hair loss generates considerable anxiety in both men and women. However, male-pattern alopecia is often more socially acceptable and has well-established treatment regimens such as finasteride. Finasteride 1 mg daily is highly effective in treating male-pattern alopecia,³ but has less clear efficacy in female

androgenetic alopecia. In 2000, a multicentre, randomized, double-blind, placebo-controlled study of postmenopausal women treated with finasteride 1 mg daily showed no improvement in increasing hair growth or slowing the progression of hair thinning.⁴ In contrast, in 2001 a noncontrolled study of 42 pre- and postmenopausal women with female-pattern hair loss and SAHA syndrome (seborrhoea, acne, hirsutism and alopecia) revealed that finasteride 2.5 mg daily effectively increased hair growth.⁵

Recently, another study supported the efficacy of medium-high doses of finasteride in the treatment of female-pattern hair loss. Approximately two-thirds of the 37 women without clinical evidence of hyperandrogenism responded well to a medium-high dose of finasteride (2.5 mg daily).¹ The authors stated that the concomitant use of the oral contraceptive drospirenone may also have contributed to the hair growth due to its antiandrogenic effect. Marked efficacy was also observed for higher doses of finasteride (1.25–5.0 mg daily) in women with normo- or hyperandrogenism in recent reports.^{2,6} As these higher doses of finasteride differ from the standard male androgenetic alopecia dose of 1.0 mg daily, an important unanswered question arises: whether androgenetic alopecia in women demonstrates a dose-dependent therapeutic response or whether some patients respond due to their relative androgen levels. Indeed, in one case report, a woman with androgenetic alopecia had limited response to finasteride 0.5 mg daily and benefited well from dutasteride, a more potent 5 α -reductase inhibitor.⁷

In our case, we cannot rule out that a lower dose of finasteride may have been effective. Further study is necessary to establish the optimal dose regimen for finasteride in female androgenetic alopecia due to androgen supplementation. Practitioners need to be aware that Hamilton type hair loss can occur in women given androgen supplementation, especially at higher doses. In our patient, androgen-induced alopecia was effectively treated with a medium-high dose of finasteride (2.5 mg daily) despite her continued androgen supplementation. Taking her surgically menopausal status into account, the testosterone adjunct to oestrogen replacement therapy may benefit our patient by reducing anxiety and depression, protecting against breast cancer⁸ and delaying Alzheimer's disease.⁹ We recommend that hormonal supplementation with androgen be appropriately reduced and maintained at a reasonable dose when iatrogenic androgenetic alopecia is found.

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Immunohistochemical characterization of elastofibroma and exclusion of *ABCC6* as a predisposing gene

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SIR, Elastofibroma (EF) is a subcutaneous fibroelastic pseudotumour that usually presents in adulthood at the lower end of the subscapular space. Microscopy reveals adipose tissue, bundles of collagen interspersed with dystrophic (globular or beaded) elastic fibres, and spindle-shaped cells dispersed in the connective tissue.¹ Multiple and familial EF have been reported, supporting the possibility of a hereditary predisposition.² We carried out an immunohistochemical study of EF to characterize further the elastic fibres and cells, and a mutation analysis of *ABCC6*, the gene responsible for pseudoxanthoma elasticum (PXE). One recent paper reported a patient with two EFs and PXE, and questioned a genetic link between both rare conditions with dystrophic elastic fibres.³

Four EF tumours from three unrelated patients were included in the study. All individuals gave informed consent for search for *ABCC6* mutations. The EF sections were incubated with antibodies against actin, desmin, vimentin, elastin,