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Diffuse face and ear hypertrichosis caused by 5% topical minoxidil in an adult woman with spontaneous resolution

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Abstract

Minoxidil is a vasodilator medication known for its ability to promote hair growth. Although it was first introduced as an oral drug to treat hypertension, minoxidil was observed to have the important sideeffect of increasing hair growth. This led to the development of a topical formulation as a 2% concentration solution for the treatment of female androgenic alopecia (AGA) and 5% for treating male AGA, which is considered as a first line U.S. Food and Drug Administration (FDA)-approved treatment for AGA in addition to oral 5-alpha-reductase inhibitor (finasteride). The mechanism by which minoxidil promotes hair growth is not fully understood but can be related to increasing blood flow owing to its vasodilator effects. Androgenic alopecia characterized by the gradual conversion of terminal hairs into vellus hairs. Alterations in the hair cycle include reduced duration of the anagen phase and increased duration of the telogen phase, resulting in shorter hairs and eventual balding. Side effects of topical minoxidil include irritant and allergic contact dermatitis, pruritus, and facial hypertrichosis, which are more often seen with the use of 5% solutions rather than 2%. Herein, we report a 24-year-old woman who developed severe ear and face hypertrichosis after using a topical 5% minoxidil solution. She later had spontaneous resolution of her hypertrichosis three months after stopping it.

Keywords: face, hypertrichosis, minoxidil, topical

Introduction

Minoxidil was first introduced as an oral medication for the treatment of resistant and severe

hypertension [1]. Coincidentally, physicians observed hair regrowth and generalized hypertrichosis in bald patients, which led to the development of a topical minoxidil formulation for treating androgenetic alopecia (AGA), first in men, and then in women [2]. Minoxidil is a potassium channel opener, causing hyperpolarization of cell membranes. It is also a vasodilator and it is speculated that minoxidil widens blood vessels and opens potassium channels, allowing more oxygen, blood, and nutrient flow to hair follicles [3]. This can also cause hair in the telogen phase to shed, usually soon to be replaced by thicker hairs in an anagen phase [2]. The most common adverse reactions of topical minoxidil are limited to irritant and allergic contact dermatitis on the scalp, in addition to face hypertrichosis [2]. Topical minoxidil is a well-known and often utilized drug in dermatological practice for the treatment of AGA. It was approved by the United States Food and Drug Administration for the management of AGA in 1988. Since its approval, minoxidil has been used off-label for the treatment of many other types of alopecia [3]. Conditions for which the use of topical minoxidil has been reported include telogen effluvium, alopecia areata, scarring alopecia, eyebrow hypotrichosis, monilethrix, and chemotherapy-induced anagen effluvium [2].

Case Synopsis

A 24-year-old woman known to have AGA was on dutasteride 0.5mg per day, and platelet-rich plasma sessions. The patient developed severe hypertrichosis over the face and ears after using minoxidil 5% solution once daily for three months



Figure 1. Hypertrichosis over face, ears, and neck.

(**Figure 1**). The patient was not on herbal or other oral preparations. The patient was advised to stop using topical minoxidil and showed complete clearance of ear hypertrichosis after three months of cessation of minoxidil. Facial hypertrichosis was subsequently successfully treated by a hair removal laser (**Figure 2**).

Case Discussion

Androgenic alopecia is the most common form of hair loss; it is characterized by a receding frontal hairline in men and diffuse patterned thinning in women, with frontal hairline retention caused by hyperactivity of 5-alpha-reductase; it frequently affects an individual's life quality [3]. Topical minoxidil stimulates new hair growth and helps to stop the loss of hair in both men and women [4].

Minoxidil is the only approved topical agent for the treatment of AGA. Minoxidil is a prodrug requiring bio-activation into minoxidil sulfate in the hair follicle outer root sheath and the enzyme that





Figure 2. Clearance of hypertrichosis over face, ears, and neck.

catalyzes this reaction in the hair follicle is sulfotransferase (SULT1A1), which has several variants [7]. The expression of SULT1A1 in the scalp varies greatly between individuals and this difference in expression explains the varied clinical response to topical minoxidil treatment. Low SULT1A1 activity occurs in approximately 60% of the population and predicts weak hair regrowth [5]. These individuals are likely to require higher concentrations of minoxidil and adjuvant treatment to compensate for low SULT1A1 activity [5].

Topically minoxidil is available in both 2% and 5% solutions and foam formulations, giving clinicians and patients more flexibility to select their preferred strength and formulation [4]. The 5% solution has demonstrated greater efficacy than the 2% solution. Foam has greater absorption compared to solution [5]. Several factors increase responsiveness to topical minoxidil including younger age, early initiation of treatment, smaller area of involvement, and increased number of terminal hairs before initiation of treatment. Usually, at least four months of continuous therapy with a minoxidil topical solution is required to show response and hair regrowth [6]. The daily dosage for men is 1ml of 5% minoxidil once or twice daily and 1ml of 2% solution or 5% of foam daily for women with a maximum dose of 2ml daily for both [7].

The most common side effect is irritant contact dermatitis with typical symptoms of pruritus and scaling. The incidence is lower with 2% compared to 5% minoxidil. Allergic contact dermatitis can also occur mainly because of propylene glycol (PG) in the solution preparation but less commonly to minoxidil itself [2]. In most patients, hypertrichosis is restricted to the face and upper limbs, possibly by inadvertent application or transfer of the product through the face [6]. In cases of generalized hypertrichosis, however, the product is unlikely to reach the entire body surface. Systemic absorption of topically applied minoxidil is minimal.

Hypertrichosis caused by minoxidil should reverse once treatment is stopped in 3-4 months. Furthermore, other treatment modalities for hypertrichosis can be considered in treating hypertrichosis caused by minoxidil including hair

removal laser, shaving, or waxing, and effornithine cream.

Eflornithine cream is the first topical prescription approved by the U.S. FDA to treat unwanted facial hair in women [8]. It irreversibly inhibits ornithine decarboxylase, an enzyme that catalyzes the ratelimiting step for follicular polyamine synthesis, which is necessary for hair growth. In clinical trials, eflornithine cream slows unwanted facial hair growth in up to 60% of women [8]. Improvement occurs gradually over 1-3 months [9]. However, its effectiveness in treating hypertrichosis caused by minoxidil has not been mentioned in previous literature.

Conclusion

Androgenetic alopecia is the most prevalent form of hair loss in men and women. Minoxidil is the first-line topical treatment for androgenetic alopecia alone or with other treatments with minimal side effects. Minoxidil 5% has greater efficacy but is more likely to cause hypertrichosis. Hypertrichosis caused by minoxidil is reversible upon stopping it.

Potential conflicts of interest

The authors declare no conflicts of interest.

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