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Frontal fibrosing alopecia with involvement of the central hair part: distribution of hair loss corresponding to areas of sunscreen application

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Abstract

Frontal fibrosing alopecia (FFA) typically presents with band-like scarring alopecia in the frontal and temporal hairline along with eyebrow loss. Although this type of hair loss is being seen with increased frequency worldwide, the etiology of the condition is unknown. Studies have suggested a potential environmental role with moisturizers and sunscreens being possible triggers. Herein, we present a 42-year-old woman with a biopsy-proven diagnosis of frontal fibrosing alopecia. In addition to the typical pattern, she also had a striking linear patch of hair loss along her central part. This was an area where she regularly applied sunscreen for many years. Although a causative role for sunscreen ingredients in the development of FFA has not been proven, the evidence accumulated thus far, including our suggestive case, substantiates the need for further study. Additionally, increasing awareness of this potential effect leading to appropriate counseling regarding cessation of possible triggers may be critical for the prevention of further hair loss.

Keywords: frontal fibrosing alopecia, sunscreen, scarring alopecia, cicatricial alopecia

Introduction

Frontal fibrosing alopecia (FFA) was first described by Australian dermatologist Steven Kossard in 1994 in a cohort of 6 post-menopausal women who presented with band-like scarring alopecia along the fontal hairline and loss of eyebrows [1]. Since the initial description, this condition has been seen with increasing frequency worldwide, though the cause for the rapid rise in cases is still unknown [2]. A recent genome-wide association study in patients with FFA identified xenobiotic, hormone processing, and immune response susceptibility loci [3]. Other studies have found that patients with FFA and other scarring alopecias exhibit an increase in the expression of the aryl hydrocarbon receptor, which is a highly sensitive environmental sensor that mediates immune responses [4]. These data along with others support the hypothesis that the pathogenesis of FFA may involve environmental insults which lead to pilosebaceous unit dysfunction, collapse of immune privilege, and a lichenoid immune response in predisposed individuals [5]. Epidemiologic studies have identified a potential link between the increased use of facial moisturizers and FFA; thus, these products have become the leading candidates in the search to identify potential environmental triggers of the disease [6,7].

Case Synopsis

The patient being presented is a 42-year-old, pre-menopausal woman who presented with loss of hair in the following areas: eyebrows, frontal/temporal hairline, central hair part, and forearms (**Figure 1**). The hair loss was gradual over two years and was not

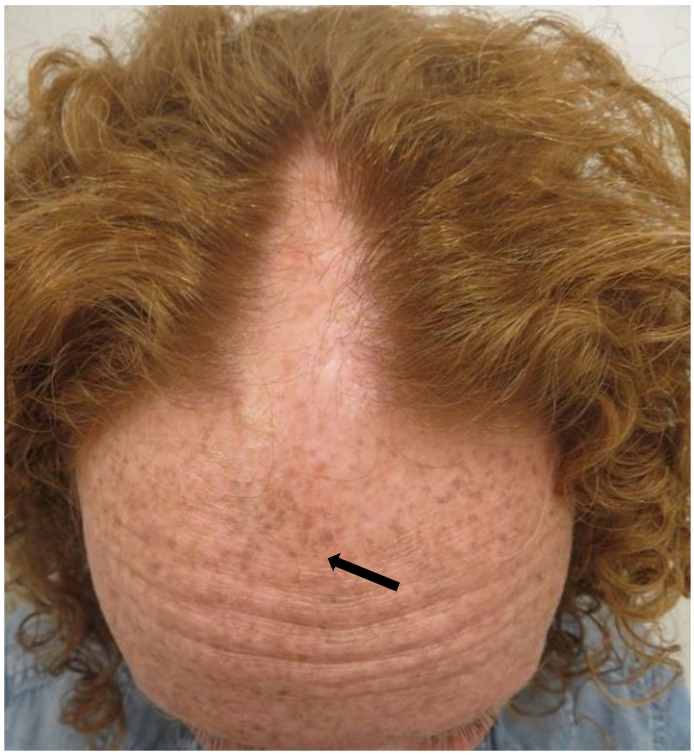

Figure 1. *Hair loss in a bandlike pattern along with hairline as well as the central hair part and eyebrows. The patient's wrinkled forehead highlights the border between her frontalis muscle and galea, the site of her anatomical former hairline, (black arrow).*

associated with any symptoms. Other than mild asthma managed with the use of an albuterol inhaler as needed, she was healthy. On questioning, she reported daily use of sunscreen on her face, arms, and central hair part for many years. Specifically, she would apply sunscreen starting from the frontal hairline to the mid-scalp along her central hair part. She denied the use of significant heat or any chemical treatments on her hair or scalp. She wore her hair down and only occasionally used a blow dryer. On examination, she had skin type 1 with extensive freckling and long, red, course, curly hair. She had alopecia with loss of follicular markings involving the fronto-temporal hairline as well as a striking linear patch along her central part. There was some perifollicular erythema at the active margin of hair loss along with a few residual terminal hairs at her former hairline. Her eyelashes were intact. She had no visible hair on her forearms. A scalp biopsy was consistent with a lymphocytic cicatricial alopecia (**Figure 2**).

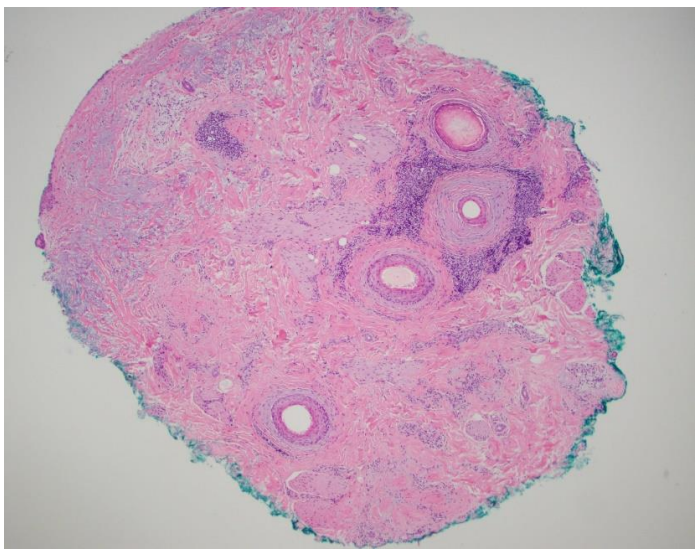

Figure 2. *Horizontal scalp biopsy. There is loss of terminal follicles and sebaceous glands, with lichenoid inflammation surrounding the isthmus of the follicle and perifollicular fibrosis. H&E, 40×.*

Case Discussion

There has been much speculation over a putative environmental trigger for FFA [8]. Proposed triggers include components of sunscreens. An initial case series demonstrated that patients with FFA were more likely to use sunscreen than controls [6]. Although this study's methodology has been the subject of criticism [9,10], several follow up studies have supported the association [7,11-12]. Our subject directly applied a sunscreen to areas that subsequently developed scarring alopecia. There is a similar report of a subject with scarring alopecia within her part following the use of a spray-on sunscreen in the area [13]. Another case report illustrates a case of FFA that positively responded to the discontinuation of sunscreen, in addition to medical management [14].

A study that employed electron microscopy found titanium oxide nanoparticles coating the hair shafts of disease-affected scalps, though a proportion of control subjects shared the finding [15]. Given that various metals have been associated with lichenoid eruptions, such as gold and cutaneous lichen planus (LP) and dental amalgams and oral LP, it is conceivable that titanium may elicit a follicular lichenoid eruption in predisposed individuals. Another study has revealed that a substantial amount of hair care products contain UV filters such

as benzophenone, benzyl salicylate, and ethylhexyl methoxycinnamate [16]. The authors then surveyed 53 of their own FFA and LPP patients, finding that all used hair care products containing these filters. There is some evidence to suggest that the active agents in chemical blocker sunscreens may have unintended biologic effects, including endocrine disrupting effects. Although the mechanism is not clear, these too could conceivably play a role in disease pathogenesis. Sunscreen preparations also contain numerous inactive ingredients, which may be relevant.

Our subject's sunscreen contained chemical blockers only, a combination of avobenzone, homosalate, octisalate, octocrylene, and oxybenzone, in addition to a number of inactive ingredients, which included multiple parabens, acrylates, and fragrance. There was no titanium dioxide present.

Conclusion

Although a causative role for sunscreen ingredients and FFA has not been proven, the evidence accumulated thus far, including our suggestive case, substantiates the need for further study. Additionally, increasing awareness and offering appropriate counseling regarding cessation of possible triggers may be critical for the prevention of further hair loss.

Potential conflicts of interest

The authors declare no conflicts of interests.

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