Dermatology Practical & Conceptual

# Frontal Fibrosing Alopecia Associated With Oral Erosive Lichen Planus: Two Locations, One Disease

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## Introduction

Frontal fibrosing alopecia (FFA) is a primary scarring alopecia that mainly affects postmenopausal women and is clinically characterized by recession of the fronto-temporal line with loss of the eyebrows [1].

## Case Presentation

An 89-year-old woman presented to our dermatology unit for a localized burning sensation in the oral cavity for 3 months, that was treated with oral acyclovir without noticeable improvement. Family history included cutaneous lichen planus in her son. Personal history included arterial hypertension, osteoporosis, and rectal resection surgery for T2N0 rectal adenocarcinoma. Physical examination revealed a few erosions, partially covered by whitish pseudo-membranes, over an erythematous base on the oral vestibule and buccal mucosa bilaterally, where whitish striae referable to Wickham striae were also observed (Figure 1A). At the same time, a symmetrical eyebrow alopecia accompanied by a bilateral fronto-temporal hairline recession with loss of visible follicular ostia was noted (Figure 1B). Trichoscopic examination showed rarefaction of follicular ostia, in the absence of active inflammatory lesions, yellow dots, or vellus hair (Figure 1C). A clinical diagnosis of frontal fibrosing alopecia was then established. General examination excluded the presence of other lesions on the skin or concomitant lichen planopilaris. The patient laboratory tests were all within reference range except for mildly increased ferritin and mild neutrophilia. A comprehensive serological autoimmunity panel gave negative results, while microscopic and culture examination of an oral cavity swab excluded the presence of bacteria or yeasts. Histopathologic examination of a biopsy specimen of the edge of an oral erosion showed a band-like lymphocytic infiltrate in the superficial chorion and at the junction with the overlying mucosa (Figure 1D), consistent with the diagnosis of oral erosive lichen planus.

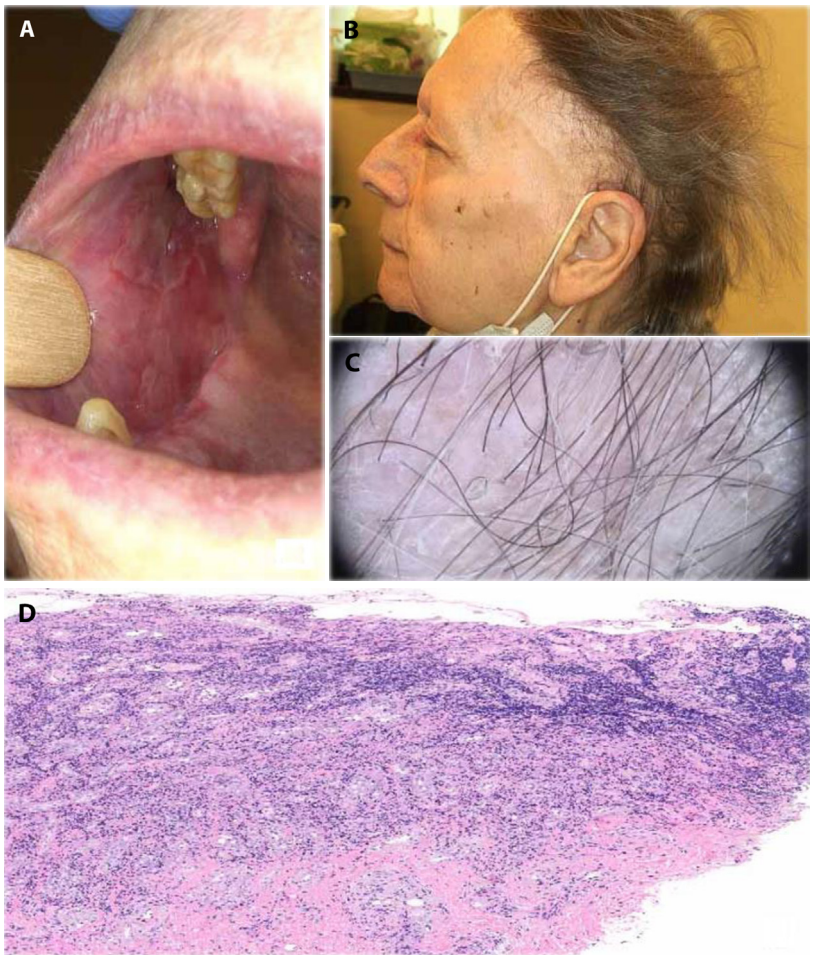


**Figure 1.** (A) Erosions over an erythematous base, partially covered by whitish pseudomembranes, located at the level of the vestibule of the mouth and buccal mucosa. (B) Symmetrical eyebrow alopecia accompanied by bilateral fronto-temporal hairline recession. (C) Trichoscopy; loss of visible follicular ostia in the absence of active inflammatory lesions, yellow dots or vellus hair. (D) Histology; band-like lymphocytic infiltrate in the superficial chorion and at the junction with the overlying mucosa.

## Conclusions

Frontal fibrosing alopecia belongs to the group of interface (lichenoid) dermatitis, being characterized histopathologically by a perifollicular lymphohistiocytic infiltrate, resulting in fibrosis and scarring [1]. These features make FFA indistinguishable histologically from lichen planopilaris, of which it is therefore considered a clinical variant [1,2]. To date, only rare cases of FFA associated with oral lichen planus (OLP) have been described [2]. Oral lichen planus is a chronic inflammatory disease also characterized by interface dermatitis on histopathology. Clinically, it can be distinguished into three main subtypes: reticular, atrophic, and erosive [3]. It has been reported a small but consistent increased risk of oral squamous cell carcinoma development among OLP patients [4]. In OLP, stimulation of cell-mediated immunity leads to activation of cytotoxic CD8+ T lymphocytes directed against cells in the basal layer of the epithelium, with an early increase in Th1-type cytokines [3]. Similarly, in FFA the interaction of genetic, hormonal, and environmental factors would cause the loss of the immune privilege of hair follicles induced by Th1-type inflammation, with up-regulation of the JAK/STAT signaling pathway and pro-fibrotic markers, increased production of IFN-γ, and subsequent activation of CD8+ lymphocytes directed against the hair follicle [1,2]. Immune dysregulation seems to be limited to the lesional skin [5].

The coexistence of OLP and FFA in our patient suggests that, although rarely associated, these two disorders could represent two manifestations of the same immune-mediated process. The presence of OLP should therefore be looked for in all patients with FFA by careful clinical inspection of the oral cavity, also taking into consideration the risk of malignant progression of oral mucosal lesions.

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