FRONTAL FIBROSING ALOPECIA ASSOCIATED WITH LICHEN PLANOPILARIS: A PEDIATRIC CASE

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Abbreviations **FFA** = frontal fibrosing alopecia; **LPP** = Lichen planopilaris.

Case report. The patient is a 12-year-old girl with an unremarkable medical history, who presented with progressive hair loss over a period of three years. Initially localized to the temporal regions, the hair loss later spread, eventually involving the eyebrows. The patient had consulted several physicians; a preliminary biopsy of the occipital region revealed end-stage fibrosis, leading to a diagnosis of pseudopelade of Brocq. She reported no similar cases in her family, history of autoimmune diseases, or stressful events preceding the onset of symptoms.

Clinical examination revealed reduced scalp hair density. In the frontal and temporal regions, recession of the hairline, loss of vellus hairs along the frontal margin, and the presence of single hairs were observed (Fig. 1, 2). Dermoscopy showed perifollicular erythema and scaling, absence of follicular ostia, and the presence of a single hair per follicular orifice (Fig. 3).

In the occipital and parietal areas, we found partially alopecic patches (Fig. 4) with a focally positive hair pull test; dermoscopy revealed atrichia, white dots, fibrosis, the appearance of a single hair per follicular orifice, perifollicular scaling, pili torti, and red areas (Fig. 5). Black dots, exclamation mark hairs, leukotrichia, and yellow dots were absent. Partial alopecia of the eyebrows was noted.

Clinical examination of the skin, mucous membranes, and nails revealed no abnormalities. Lichen planopilaris and alopecia areata were considered in the differential diagnosis. A repeated biopsy from the advancing inflamed margin, guided by dermoscopy, revealed a perifollicular lymphocytic infiltrate

Fig. 1

Fig. 2

Fig. 3

Fig. 1, 2, 3: Hairline recession, loss of vellus hairs in the frontotemporal area, and isolated hairs (Fig. 1, 2). Dermoscopy (Fig. 3) show s single hair emergence per follicular opening, perifollicular scaling (red triangle), absence of follicular ostia (blue star), white dots (blue arrow), and fibrosis (black arrow).

Fig. 4

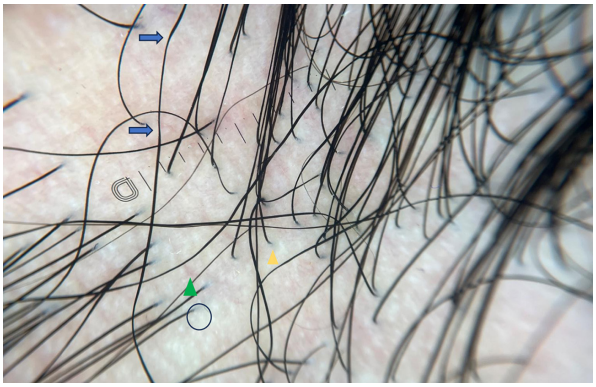

Fig. 5

Fig. 4, 5: Patches of alopecia (Fig. 4). Dermoscopy shows single hairs per orifice, thick and irregular linear vessels (green triangle), pili torti (blue arrow), and white dots (blue circle).

involving the isthmus and infundibulum, with interface dermatitis and basal cell vacuolization, consistent with lichen planopilaris. Therefore, a diagnosis of frontal fibrosing alopecia associated with lichen planopilaris was established.

The patient was treated with oral corticosteroids (40 mg/day for two weeks with a gradual taper), doxycycline 100 mg/day, monthly intralesional betamethasone injections for six months, and vitamin D supplementation. At the six-month follow-up, the patient reported clinical improvement, with complete regrowth of the eyebrows and partial hair regrowth (Fig. 6, 7). The hair pull test was negative, and trichoscopy showed vellus hairs and focally regrowing hairs.

Discussion. Lichen planopilaris and infantile androgenetic alopecia are primary cicatricial alopecias that predominantly affect middle-aged adults and have only been rarely reported in children. Infantile lichen planus accounts for approximately 1% to 4% of all cases, and follicular involvement remains particularly rare in this age group (1). Consequently, both lichen planopilaris and infantile androgenetic alopecia are poorly characterized, and most data derive from isolated case reports or small case series (5).

These two conditions share a central mechanism involving an autoimmune reaction mediated by CD8+ cytotoxic T lymphocytes, which target the hair follicle. These inflammatory cells primarily surround the upper part of the follicle, which houses the epithelial stem cells. This leads to a loss of immune privilege and triggers the apoptosis of follicular stem cells, preventing follicle regeneration and ultimately resulting in permanent scarring and hair loss (6).

Other factors are involved in frontal fibrosing alopecia (FFA), particularly a hormonal component that is widely documented and partly explains its strong predisposition in postmenopausal women. Familial cases and genetic studies suggest a hereditary or epigenetic component, although the specific genes have yet to be identified. Finally, environmental triggers, primarily cosmetic products and ultraviolet radiation exposure, are also involved, although their exact role has not been definitively established (7).

Frontal fibrosing alopecia is considered a clinical subvariant of lichen planopilaris (LPP). Indeed, they share histological similarities. The initial active phases feature a perifollicular lichenoid inflammatory infiltrate involving the isthmus and infundibulum, whereas later stages are characterized by

Fig. 6

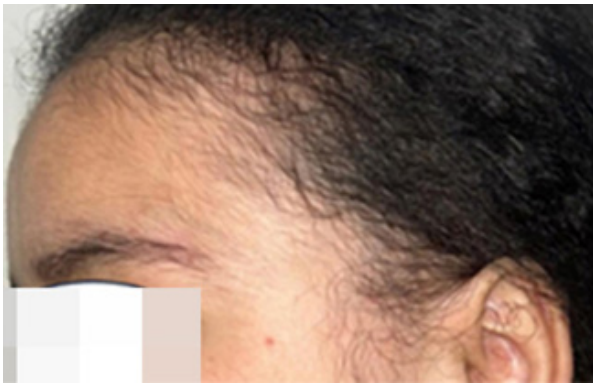

Fig. 7

Fig. 6, 7: Six months later, hair regrowth at the hairline.

perifollicular fibrosis with alopecia and follicular scarring that replaces the pilosebaceous units (8). Conversely, in alopecia areata, the inflammatory infiltrate affects the bulb, which is located in the lower third of the follicle (8). This difference helps to distinguish alopecia areata from frontal fibrosing alopecia, as illustrated in our case.

Clinically, the classic form of LPP most commonly affects the vertex, but any region of the scalp can be involved. The initial lesions are characterized by perifollicular violaceous erythema and keratotic follicular plugs (2). However, frontal fibrosing alopecia is characterized by a progressive recession of the frontotemporal hairline, frequently associated with eyebrow alopecia (9).

Dermoscopy plays a fundamental role in distinguishing LPP and FFA from other causes of pediatric alopecia. In the present case, trichoscopy was instrumental in ruling out alopecia areata and trichotillomania. Consistent with our case, the absence of leukotrichia, black dots, and exclamation mark hairs argued against alopecia areata, whereas the lack of flame hairs, hair powder, and hook hairs helped to exclude trichotillomania (10, 11, 12).

The management of FFA and LPP in children represents both a diagnostic and therapeutic challenge. Given its rarity, there are no standardized treatment guidelines, and therapeutic decisions are often extrapolated from adult data or limited pediatric studies (5, 12). The primary goal of treatment is to suppress follicular inflammation and prevent irreversible cicatricial alopecia.

First-line therapy is based on topical anti-inflammatory agents, including high- or ultra-high-potency topical corticosteroids, topical calcineurin inhibitors, and intralesional corticosteroid injections, most frequently triamcinolone acetonide. When triamcinolone is not available, as in our case, intralesional betamethasone dipropionate can be considered an alternative. Topical minoxidil can be used as an adjunctive therapy to improve hair density (5). Systemic treatments are reserved for extensive or progressive forms of the disease and may include oral corticosteroids, methotrexate, or doxycycline, which can be prescribed in children aged 8 years and older. In refractory cases, the use of immunosuppressants or biologics such as cyclosporine, mycophenolate mofetil, or tocilizumab has been reported (12).

Conclusion. Frontal fibrosing alopecia and lichen planopilaris are exceptional causes of cicatricial alopecia in children, often posing diagnostic and therapeutic challenges. This case highlights the importance of considering frontal fibrosing alopecia and lichen planopilaris in the differential diagnosis of pediatric cicatricial alopecia and emphasizes the value of dermoscopy in guiding diagnosis and management.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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