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Fibrosing alopecia in a pattern distribution after hair transplantation: A case report and literature review

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Abstract

Patterned fibrosing alopecia is a form of scarring alopecia that shares clinical, tracheoscopy, and histopathological features with lichen panoplies and androgenetic alopecia. Its onset has been observed in patients following procedures such as hair transplants, facelifts, and other medical-surgical interventions. The Koebner phenomenon, along with alterations in the peripheral follicular immune response, is thought to trigger this condition. Literature reports a highly variable post-transplant evolution time, ranging from 3 months to 9 years. Clinically, it presents with hair loss in areas sensitive to androgens, and tracheoscopy reveals findings such as erythema, peripolar scales, reduced follicular openings, and follicular miniaturization. The therapeutic approach remains undefined, with various strategies being employed, including immunomodulators, anti-inflammatory steroids, antiandrogens, and hair growth promoters. New technologies are also currently under investigation. In this context, we present the case of a 28-year-old male diagnosed with patterned fibrosing alopecia following a Follicular Unit Extraction (FUE) hair transplant, confirmed through tracheoscopy and histopathology. The patient was treated with immunomodulatory therapy, intralesional steroids, antiandrogens, hair growth promoters, and 675 nm laser therapy, resulting in modest clinical improvement.

Keywords: Fibrosing alopecia; Scarring; Idiopathic; Hair transplant; Laser.

Introduction

Patterned fibrosing alopecia was first described in 2000 by Trueb and Zinkernagel as a scarring alopecia that shares characteristics of lichen panoplies and androgenetic alopecia [1]. It is characterized by chronic inflammation that leads to perifollicular fibrosis and follicular destruction [2]. Although the pathophysiological mechanism is not yet fully understood, autoimmune factors have been implicated, including the activation of T lymphocytes and apoptosis of follicular epithelial cells [3]. In some cases, an inflammatory response may be triggered by external insults such as hair transplants and other surgical procedures, suggesting a possible link to the Koebner phenomenon [4]. Due to the rarity of this condition and the potential for misdiagnosis with other forms of scarring alopecia, it is critical to consider it in patients with alopecia after a hair transplant, particularly those with a history of suspected scalp psoriasis or seborrheic dermatitis [5]. In this context, we present a clinical case of a 28-year-old male diagnosed with patterned fibrosing alopecia following a Follicular Unit Extraction (FUE) hair transplant, providing further insight into the diagnostic process and management challenges of this condition. **Citation:** Sánchez-Dueñas LE, Gastelum-Ibarra AE, Iribe-Martínez JP, Aguirre-Sánchez JR, Aviña-Padilla N. Fibrosing alopecia in a pattern distribution after hair transplantation: A case report and literature review. J Clin Images Med Case Rep. 2025; 6(5): 3585.

Case history

A 28-year-old male from Michoacán, Mexico, with no relevant medical history, was referred to our clinic by his private dermatologist with a diagnosis of seborrheic dermatitis and acne vulgaris. He had been treated with isotretinoin for one year. During the initial consultation, a localized dermatosis on the scalp was observed, affecting the frontal and vertex regions. The lesions presented as pseudo alopecic areas accompanied by erythema and scaling (Figure 1A, 1B). Tracheoscopy examination revealed perifollicular and interfollicular scaling, erythema, brown peripolar halos, and the presence of pinpoint and glomerular vessels (Figure 2A). Based on these findings, the coexistence of seborrheic dermatitis, telogen effluvium, and androgenetic alopecia was suspected. Initial management included anti-inflammatory agents, antiandrogens, and hair growth stimulants. At follow-up, the patient reported having undergone a follicular unit extraction (FUE) hair transplant in January 2023, after which he experienced significant hair shedding. A Trichogramma demonstrated a predominance of hairs

in the anagen phase (Figure 2B). Due to the clinical suspicion of scalp psoriasis, a skin biopsy was performed. Histopathological analysis revealed focal hypogranulosis of the epidermis, mild and irregular acanthosis, a perifollicular lymph histiocytic infiltrate around the upper isthmus, perifollicular fibrosis, and sebaceous gland hypoplasia (Figures 3 and 4). Laboratory evaluations to rule out thyroid dysfunction and insulin resistance were within normal limits. Considering the clinical presentation, tracheoscopy findings, and histopathology, a diagnosis of patterned fibrosing alopecia following hair transplantation was established. Treatment was initiated with hydroxychloroguine (400 mg daily for 2 months, then 200 mg daily for 4 months), dutasteride (0.5 mg daily for 6 months), oral minoxidil (5 mg daily for 6 months), topical calcipotriol/betamethasone (twice weekly), intralesional triamcinolone injections (12 mg monthly for 3 sessions), exosome therapy, and monthly sessions of 675 nm laser therapy. Despite treatment, the patient continued to experience substantial hair loss with only moderate clinical improvement. Consequently, second-line immunomodulatory therapy with methotrexate was initiated.



Figure 1: (A) Scalp dermatosis with diffuse involvement, presenting as pseudoalopecic plaques accompanied by perifollicular erythema and scaling. **(B)** Frontal view revealing frontoparietal hairline regression with mild erythema and fine perifollicular scaling.



Figure 2: (A) Trichoscopic image demonstrating hair regrowth, isolated follicular units, interfollicular erythema and scaling, brown peripilar halo, and follicular miniaturization. **(B)** Trichogram showing numerous anagenphase hairs with characteristic pigmented "golf club"-shaped bulbs.



Figure 3: Histopathological image. The epidermis displays focal hypogranulosis, mild and irregular acanthosis, and exudate within the stratum corneum. In the mid-dermis, there is a perifollicular inflammatory infiltrate predominantly composed of lymphocytes, localized around the upper isthmus. Associated findings include perifollicular fibrosis, terminal hair follicles mainly in the anagen phase, and marked sebaceous gland hypoplasia.

 Table 1: Comparative summary of case series described in literature.



Figure 4: High-power histopathological image (40x) showing a perifollicular lymphocytic infiltrate and concentric fibrosis surrounding the affected hair follicles.

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Summary of reported cases of post-transplant patterned scarring alopecia					
Study	Number of patients	Age	Symptom onset post- transplant	Symptoms	Clinical and trichoscopic findings
Donovan et al 2012 9 [8]	17 patients (15 M, 2 F)	30-67 years	4 a 36 months	Pruritus and asymptomatic	Erythema, peripilar scaling, loss of follicular openings. LPP 70%[U3]
Banka et al 2014 [13]	26 patients (17 M, 9 F)	22-65 years	History of seborrheic dermatitis	Asymptomatic or mild pruritus	>20% variability in frontal, parietal or vertex regions, erythema, 2–4 hairs emerging from follicular opening, peripilar and interfollicular scaling, absence of follicular openings
Chiang et al 2012 [9]	10 patients (4 M, 3 F)	34-63 years	3 months to 9 years / 2 (face lift) AFF	Pruritus	Diffuse hair loss, erythema, scaling, and loss of follicular openings
Rocha Crisostomo et als 2009 [14]	2 patients (2 M)	50 years, 46 years	6 years, 2 years	Asymptomatic	Diffuse hair loss in recipient area, erythema and perifollicular scaling

Discussion

Fibrosing Alopecia in a Pattern Distribution (FAPD) is a form of scarring alopecia that remains underrecognized within the medical community. Its distribution pattern often mimics that of androgenetic alopecia [6]. Several reports have described its onset following hair transplantation and facial surgery, suggesting that the Koebner phenomenon and an autoimmune response targeting hair follicles-characterized by perifollicular lichenoid inflammation and progressive fibrosis-may contribute to its pathogenesis [7]. Donovan et al. (2012) documented 17 cases of lichen panoplies following hair transplantation, with a mean onset of 11 months post-procedure [8]. Similarly, Chiang et al. [9] reported the development of lichen panoplies between 3 months and 9 years after surgery (Table 1). In line with existing literature, our patient, who had a history of seborrheic dermatitis and androgenetic alopecia, developed scarring alopecia one year after undergoing FUE. The tracheoscopy findings observed-erythema, peripolar scaling, follicular miniaturization, and loss of follicular openings-were consistent with those described by Griggs et al. in cases of FAPD [10]. There is currently no standardized treatment for this condition. Therapeutic goals focus on reducing inflammation, halting follicular miniaturization, and promoting hair regrowth. The literature describes the use of immunomodulators such as hydroxychloroquine and JAK inhibitors, as noted by Tekin et al [11]. In our patient, antiandrogen therapy (finasteride, dutasteride) was combined with complementary approaches, including 675 nm laser therapy, to target the androgenetic component [12]. Further studies are required to clarify the pathogenesis, define clinical and tracheoscopy features, and establish comparative trials that may optimize therapeutic strategies and improve patient outcomes [9].

Conclusion

Fibrosing alopecia in a pattern distribution is a scarcely recognized form of scarring alopecia that primarily affects androgen-dependent areas. Its potential association with medicalsurgical procedures such as hair transplantation and facial lifting has been proposed, suggesting the involvement of the Koebner phenomenon and a perifollicular autoimmune response. Differential diagnoses for anagen effluvium should include scalp psoriasis, lichen planopilaris, and alopecia areata. Although there is no standardized treatment protocol, therapeutic approaches have included corticosteroids, immunomodulators, antiandrogens, and laser therapy. Further research is essential to better understand its pathogenesis and to develop more effective treatment strategies.

Declarations

Competing interests: The authors declare no competing interest.

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