Hair loss due to lichen planopilaris after hair transplantation: a report of two cases and a literature review *
Perda pilosa por líquen plano pilar após transplante capilar: relato de dois casos e revisão da literatura

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Abstract: Androgenetic alopecia is often treated by follicular unit transplantation, a technique that involves minimal risk of hair loss because of the more resistant nature of the donor area. Lichen planopilaris is a cicatricial alopecia that causes permanent destruction of hair follicles. We report two cases of post-transplantation lesions compatible with lichen planopilaris in both recipient and donor areas. The quality of the hair follicles in the donor area was apparently compromised by lichen planopilaris, the probable cause of hair loss. Similar reports are rare. When lichen planopilaris is suspected, a biopsy of the scalp must be performed to avoid transplantation during disease activity.

Keywords: Alopecia; Lichen planus; Transplantation; Transplantation, autologous

INTRODUCTION
Alopecia is a chronic dermatological condition in which there is complete or partial hair loss from the scalp. Other parts of the body may also be involved. The physiopathogenesis is not fully understood, but the condition involves an inflammatory process that is the result of a combination of genetic and environmental influences. The most common cause of hair loss is androgenetic alopecia (AGA), which affects approximately 50% of 50-year-old men and 20% to 50% of women of the same age.1,2

For permanent baldness, surgical treatment in the form of follicular unit transplantation is indicated.3,4 This involves removing a strip of scalp from the lateral and occipital region of the head, an area that is generally not affected by androgenetic alopecia.3 Follicular units containing one to four follicles are then prepared and implanted in the bald area.3 After they have grown, the transplanted hairs no longer fall out as they retain the characteristics of the donor area, where they are resistant to the effects of AGA.5

The term cicatricial alopecia covers a group of different skin disorders characterized by permanent destruction of the hair follicle and residual fibrosis.6 Many etiologies can be implicated in this condition, including traumas, such as burns, and congenital, inflammatory and infectious disorders.6,7 The various
etologies covered by the term cicatricial alopecia can be classified into stable and unstable forms. The latter are characterized by their tendency to progress and recur. They include alopecias due to inflammatory causes, 40% of which are accounted for by lichen planopilaris (LPP). Cicatricial alopecia can affect the whole scalp, including the donor area from where hairs are removed for transplantation.

The aim of this study is to describe two cases of loss of transplanted hair, probably as a result of LPP.

**CASE REPORTS**

Patient 1: A 50-year-old male presented with a complaint of hair loss in the recipient area following a hair transplant carried out in another service six years previously. Examination revealed diffuse thinning and hair loss on the top of the head and the vertex (Figure 1). Diffuse and coalescing areas of hair loss suggestive of cicatricial alopecia were observed on the lateral and occipital region of the head (Figure 2). A new hair transplant was contraindicated in this case because there was extensive involvement of the donor area and the patient’s expectations were incompatible with the result that could be achieved. This patient was not followed up.

Patient 2: A 46-year-old male patient who had had a hair transplant 2 years previously in another service and complained, like the first patient, of partial loss of the transplanted hair. On examination, areas of diffuse alopecia were observed on the top of the head and vertex (Figure 3). Areas of diffuse thinning were observed on the rear and sides of the head that were suggestive of cicatricial alopecia and perifollicular erythema in some of the lesions, indicating disease activity.

A fragment of skin from the scalp in the posterior-lateral region of patient 2’s head was submitted for histopathological examination; the results of this were suggestive of lichen planopilaris (Figure 5). The patient was treated with intralesional triamcinolone. The lesions stabilized and the peripheral erythema disappeared. A new transplant has not yet been recommended, but the patient is still being followed up.

**DISCUSSION**

Initially described by Pringle in 1985, lichen planopilaris (LPP) is a skin disorder affecting hairy parts of the body. It involves a lymphocytic inflammatory process that can destroy hair follicles by replacing them with a fibrous tissue. The condition is often chronic and recurring; while it is still of uncertain etiology, autoimmune processes are probably involved in its pathogenesis. It can be divided into three variants: the classic form of LPP, frontal fibrosing alopecia and Graham-Little syndrome. Although they preferentially affect different age groups, all three forms share the same inflammatory process characterized by erythematous papules. LPP is more common in women, who account for 60% to 90% of cases, and onset is usually in the fifth to seventh decade of life.

As in the cases described above, lesions frequently involve the vertex, although any part of the scalp can be affected. Classical early-onset lesions are characterized by violet-colored follicular erythema and acuminate keratotic plugs, preferentially located on the edge of the area affected by alopecia, where the condition is expanding. The inflammatory process tends to cease spontaneously. After the hair has fallen out and the inflammation has been resolved, the lesions are replaced by atrophic scars, with permanent loss of follicles. This corresponds to the final stage of any cicatricial alopecia. The course of the disease is unpredictable and has no apparent resolution.
However, there are no long-term studies in the literature that shed light on the natural course of the disease.

Differential diagnosis is made with discoid lupus erythematosus, pseudopelade of Brocq and alopecia areata. These conditions can be distinguished by histopathological examination of biopsy specimens, especially in the initial, active stage of the disease. Histopathology reveals inflammation with hypergranulosis, hyperkeratosis, acanthosis, degeneration of basal keratinocytes and the basal layer in half of LPP cases. A subepidermal infiltrate is usually present around the follicles between the infundibulum and isthmus but not the inferior unit (unlike in alopecia areata). Colloid bodies can be observed in the basal layer; these are degenerated keratinocytes that stain pink with eosin. The same histopathological pattern was observed in patient 2 (Figure 5). Discoid lupus erythematosus can be differentiated from LPP by the predominantly central pattern of the lesions and by perivascular lymphocytic infiltrate in the deep and superficial dermis as well as in other attached structures. When LPP is not expanding and perifollicular inflammation and hyperkeratosis are absent, the condition cannot be distinguished from pseudopelade of Brocq, which some authors consider a final stage of LPP rather than a distinct entity.

**FIGURE 3:** Patient 2: Recipient area with diffuse thinning

**FIGURE 4:** Patient 2: Donor area with diffuse thinning suggestive of cicatricial alopecia

**FIGURE 5:** Patient 2: Histological section of scalp (hematoxylin-eosin), showing follicular hyperkeratosis, vacuolated cells in the basal layer, dense interface infiltrate and infiltrate around the hair follicles, as well as loss of follicles. A and B: lower magnification (10x); C and D higher magnification (40x)
Treatment of LPP is essentially clinical and is intended to reduce the severity of the symptoms, such as discomfort, itching and hair loss, and to prevent the inflammation spreading. First-line therapy continues to be intralesional injection of corticosteroids such as triamcinolone, which aims to reduce the inflammatory process and allows the progress of the alopecia to be halted, as in patient 2, whose lesions stabilized. Systemic treatments have been used in cases that do not respond to this initial treatment. Once the inflammation has been resolved and the alopecia has stabilized, surgical treatment such as scalp reduction or hair transplantation can be considered for these patients. There is little information on hair transplantation in patients with LPP in the literature. However, some authors suggest, based on empirical findings, that hair transplantation should not be indicated until at least two years have passed without any disease activity and that the patient should be warned that graft integration might be reduced by between 60% and 90%. It should be borne in mind that the possible systemic and autoimmune nature of LPP may interfere with the final outcome of the hair transplantation.

Reports of LPP developing after hair transplantation as in our case are scarce in the literature. Kossard et al. report the case of a 75-year-old man whose hair only began to thin at the front of his scalp 10 years after the last hair transplantation although he had undergone many procedures prior to that.

Drugs, infections, genetic factors and immunologic abnormalities have been put forward as factors that may trigger LPP. In the patients described, the disease apparently spread to the donor area, which probably led to the transplanted hair falling out. The question remains as to whether hair transplantation may or may not have helped trigger LPP.

Follicle loss following hair transplantation can occur immediately or, in the case of androgenic alopecia, gradually. This contrasts with the late-onset, progressive process in the patients described here. If LPP is present, it becomes even more important that the patient undergo a detailed clinical and laboratory evaluation, with particular emphasis on the donor area. Entities such as cicatrical alopecias must always be considered when there is thinning in the donor area or thinning that does not follow the pattern of androgenic alopecia. Hair implantation is a permanent procedure, and an unsuccessful outcome can be a source of dissatisfaction to the patient for various reasons. It is essential to examine the donor area when planning a hair transplant. If LPP is suspected, a scalp biopsy should be performed and a hair transplant should be avoided when the disease is active since the transplanted hairs may fall out later, as in the two cases described here.

REFERENCES

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