Localized Telogen Effluvium of the Donor Area After Hair Transplant Surgery in 12 Patients

Hair transplant restoration (HTR) is a minimally invasive surgical intervention. Its complications are generally mild and are classified into recipient area, donor area, and general complications.[1] The appearance of a diffuse telogen effluvium (TE), also called post-transplant shock loss, has been described in a large number of patients, affecting the nontransplanted follicular units (FU) in both in the donor and recipient areas between 2 and 4 months after the surgery.[2,3] Telogen effluvium is defined as a generalized loss of hair density, based on the simultaneous entry and fall of numerous FU in the telogen phase, related to the surgery-induced stress. Literature describing localized TE of the donor area (LTEDA)[4] is scarce. We describe the clinical characteristics and evolution of a case series with post-transplantation LTEDA.

Overall, 12 patients with a mean age of 39.8 (range, 26–52) years who underwent HTR and developed LTEDA were included. The epidemiological and clinical features are shown in **Supplemental Digital Content 1** (see **Table S1**, http://links.lww.com/DSS/A526). The presurgery diagnosis was androgenetic alopecia in 11 of the 12 patients. The follicular unit extraction technique was primarily used, and an average of 2,529 FU were transplanted. Localized TE started postsurgery after an average of 2 months, causing partially alopecic circumscribed areas at different regions of the donor area, with an average patch size of 7.2 cm and high variability among patients (Figure 1). In follicular unit strip surgeries (FUSS), alopecic involvement was distributed in all cases around the linear strip extraction scar. The pull test was negative in all patients. Trichoscopy was performed in 4 patients, with similar findings as follows: presence of red dots, black dots, and short dystrophic broken hairs (Figure 2). All patients refused biopsy. No specific treatment was administered in hair loss areas. In all cases, complete regrowth was observed after a mean of 4.5 months (range 3–6.5) after surgery.

To the best of our knowledge, LTEDA has been previously reported in only 2 patients.[4] The physiopathological mechanism is probably the same as conventional TE in which a trigger, such as the surgery, causes the early end of the anagen phase in a group of FU that then transition to the catagen and telogen phase, with consequent fall after 10 to 14 weeks.[5] The circumscribed and nondiffuse involvement in the presented cases could be justified by a more intense local trauma during the extraction of FU in this area and by the localized use of a large volume of tumescent anesthesia in certain zones, causing a higher local pressure. The appearance of alopecic patches around the suture points of the removed strip in the FUSS technique could support these hypotheses. Typically, the clinical presentation of the circumscribed LTEDA has a well-defined temporal sequence of appearance as follows: hair loss from the second month with a complete self-regrowth during the next months. An over-extraction of FU would produce a localized alopecic area in the donor area just after the surgery and a complete regrowth would not be observed over the course of time. When using the FUSS technique, a differential diagnosis with the surgical scar dehiscence should be performed. Self-recovery would not be observed in the latter case. An additional differential diagnosis is alopecia areata. Some of the trichoscopic findings described in LTEDA may overlap with those typically defined in alopecia areata and in trichotillomania or trichoteiromania, which share black dots or broken hairs. However, in LTEDA, the pull test is negative, and the alopecia patches are limited to the donor area and present a spontaneous regrowth in the following months. In these cases, despite not having a histological confirmation, the previous history of HTR with, the evolution with self-recovery of the alopecic patches and clinical findings led us to the diagnosis of LTEDA. There is not enough evidence showing that any treatment accelerates hair regrowth in LTEDA. However, because of the known influence of the drug minoxidil on the follicular cycle prolonging the anagen phase, it could facilitate an earlier re-entry of the affected follicles in this phase when administered both orally and topically. The regrowth of LTEDA is complete in all cases after a few months, having the same evolution of diffuse forms of posthair transplant TE. Importantly, the small sample size and the absence of histological confirmation are limitations of this study.

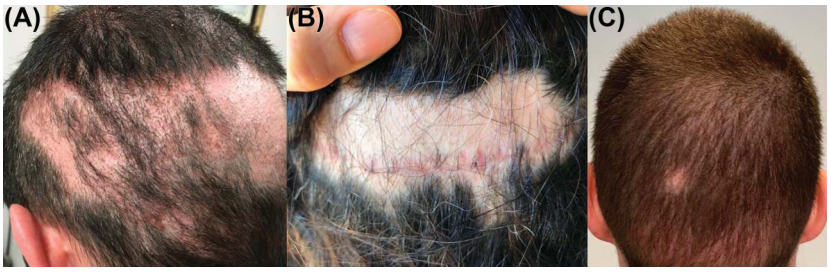

Figure 1. Clinical presentations of localized telogen effluvium of the donor area, with extensive affected areas in (A) after FUE technique or after FUSS technique around the scar (B). In case (C) small and localized occipital area affected after FUE technique. FUE, follicular unit extraction; FUSS, follicular unit strip surgeries.

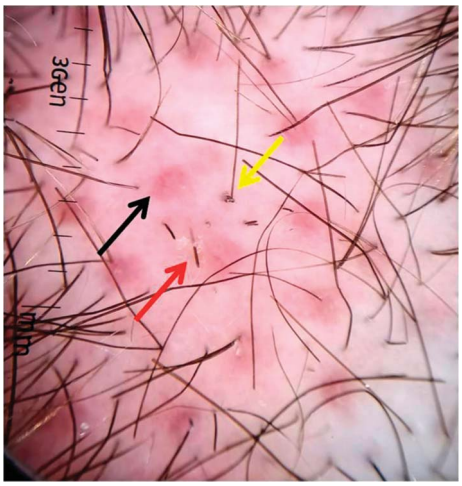

Figure 2. Trichoscopic findings in different patients, with red dots (black arrow), black dots (yellow arrow), and dystrophic short hairs (red arrow).

Localized TE of the donor area is an under-recognized entity after hair transplant restoration. The appearance of localized patches in the donor area 2 months after the surgery that can spontaneously regrow in the next months is highly suggestive of LTEDA. The prognosis is excellent, so the knowledge of this entity is primarily relevant for the hair surgeon to disseminate relevant complications to the patient.

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Ulerythema Ophryogenes Mimicking Frontal Fibrosing Alopecia

Ulerythema ophryogenes (UO), also known as keratosis pilaris atrophicans (KPA) faciei, is an atrophic variant of KP characterized by inflammatory follicular-based papules of the eyebrows that may result in scarring, atrophy, and alopecia.[1] In this article, we report a man with UO who presented with bilateral sideburn and eyebrow loss. To the best of our knowledge, the irreversible loss of sideburns and eyebrows has only been described in frontal fibrosing alopecia (FFA), a primary lymphocytic cicatricial alopecia considered a variant of lichen planopilaris (LPP) and characterized by progressive frontotemporal hairline recession.[2]

A 41-year-old Caucasian man presented with a 34-year history of loss of eyebrows in the absence of any symptoms. In his early years, the eyebrow areas were red and scaly, and he was diagnosed with psoriasis. Four months previously, he had been seen in another clinic and diagnosed with alopecia areata. He was treated with intralesional triamcinolone injections twice but did not experience any regrowth. He had never grown sideburns but denied any loss of scalp or body hair. He was otherwise healthy and did not take any regular medication. There was no known family history of alopecia areata or other hair loss conditions.

Physical examination revealed loss of the lateral eyebrows (Figure 1) with normal eyelashes. There was reduced facial hair on the cheeks and absence of sideburns; however, a few lonely hairs were noted on close inspection (Figure 2). On the forearms, there were spiny, keratotic, follicular papules with sparse hair. He had mild bitemporal recession but no reduction of hair density on the vertex. Dermoscopic examination of the eyebrows revealed terminal and vellus hairs, loss of follicular ostia but no perifollicular erythema or scaling. There was no scarring or perifollicular erythema or hyperkeratosis along the anterior hairline. A biopsy from the eyebrow showed a mild-to-moderate perifollicular lymphocytic infiltrate in the superficial dermis, numerous fibrous stellae, hair follicles of varying sizes, mild perifollicular fibrosis, and superficial vascular ectasia.