Inflammatory Alopecia: A Hidden Cause of an Announced Surgical Failure. 
The Importance of Koebner Phenomenon in Trichology

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Autographs of autologous hair is now an established practice for 
the treatment of people suffering from male and female Androgenetic 
Alopecia with an inadequate response to medical therapies or with 
no wish of getting any treatment at all. This is a short commentary to 
stimulate the attention of specialists who, during their professional 
activity, have undoubtedly noticed the existence of patients with 
poor results or even worsening of their clinical situation after surgical 
treatment. This would happen in 5% or even more of the cases followed 
in a long follow up, according to a short informal survey I conducted 
among well-known italian colleagues experts in Hair transplantation. 
Surely these data should be definitely deepened.

Another field of application of this practice is represented by forms of 
scarring post-traumatic or post-inflammatory alopecia, but only 
where the underlying disease is settled - according to authoritative 
opinions - for at least 2-3 years [1]. Despite these precautions, the 
observation in some cases of reactivation of the disease after surgical 
excision or autographs, advises us to be extremely cautious in the 
treatment of patients with Discoid Lupus Erythematosus (DLE), 
Lichen Plano Pilaris (LPP) and Pseudopelade of Brocq (PP) (Figures 1,2,3). We recently suggested that this reactivation of the inflammatory 
process could be attributed to a true Koebner Phenomenon (KP) in 
in which "Koebnerization" is inseparable from pathogenesis, treatment 
and prognosis of the original disease [2].

In 1872 Heinrich Koebner described a phenomenon he had 
observed which consisted of the following: physical trauma induced 
the formation of a characteristic of psoriatic lesions in a patient affected 
by psoriasis. Since then the term "Koebner Phenomenon" or "reactive 
isomorphism" has been utilized by the clinicians in other diseases which 
likewise present the formation of lesions of the underlying disease 
after trauma in the active forms of the diseases, where, a subclinical 
inflammation could be present also in normal appearing cutis [3].

We signalled Koebnerization in cases of LPP, DLE and also Alopicia 
Areata (AA) of the scalp [4]. We observed the classical reactivation of 
DLE after surgical exeresis of the bald patch [5], moreover we noted 
that the pathology was reactivated and widespread 3 weeks after a single 
session of cryotherapy. This also occurred after autologous hair grafting 
in micrografts, 4 weeks after transplant. In AA patches of alopecia may 
appear after obvious trauma such as being hit, burns, freezing, surgery, 
but also after prolonged microtrauma like in the cases we observed 
due to friction by clothing, glasses and buckles (Figure 4). As known, 
lichen ruber planus (LRP) is rarely found on the scalp, but typically 
presents the KP. Therefore LPP seems to have similar characteristics to 
LRP. Classically, the KP involves the epidermis, whereas in these cases 
damage was limited to the adnexal appendages.

The pathogenesis of KP is not entirely known. Toruniowa 
and Jablonska considered the mast cell (MC) as the element that 
triggers the KP in psoriasis [6]. We suggested that the MC could 
be a key cell also in the cases observed by us. In fact, in the initial 
phases of spontaneous and koebnerized AA and LPP there was an 
evident intense degranulation of mast cells (Figure 5), even when the 
mononuclear infiltrate was scarce. The MC could initiate the lesions on 
the basis of its high sensitivity to changes in temperature, concentration 
of the electrolytes, variations of pressure and even alterations of 
electromagnetic fields, besides the well-known immunological 
activation via IgE, immunocomplexes, cytokines, and neuromediators 
such as P and Corticotropin-Releasing Hormone CRH [7].

The identification of this phenomenon may explain why some 
treatments are ineffectives or even may worsen the clinical picture if 
used in the active phases of these hair diseases.

A frequent problem in Hair transplantation is the non-recognition 
of some forms of inflammatory alopecia such as 'Frontal Fibrosing 
Alopecia (FFA)” or Fibrosing Alopecia in a Pattern Distribution 
(FAPD) (clinical less inflammatory variants of LPP) [8] (Figures 6,7,8), 
easily confused with the classic Androgenetic Alopecia (AGA) and 
so aggravated by surgical treatments by the KP for the same reason 
described above. Another option that leads almost exclusively to a 
failure intervention is the lack of recognition of specific clinical aspects 
of AA: "Androgenetic-like AA”, "AA incognita”, and some forms of 
Ophisyasis (Figures 9,10,11).

All these forms can sometimes elude even the diagnosis of a 
specialist dermatologist, who has often to use imaging techniques such 
as dermoscopy (Figure 12) or histopathology to confirm the diagnosis 
[9].

Unfortunately, as previously stated, the same and apparent classic 
AGA sometimes can have disappointing results after a suitable surgical 
treatment. These findings could be explained on the basis of a Koebner- 
like Phenomenon for the presence of a subclinical perifollicular micro-
inflammation, consisting of a lymphomononuclear infiltrate mostly 
localized around the Isthmus of the hair follicle, with increase in 
number and signs of mast cell activation [10-12] in about half of these 
subjects-male and female.

This follicular micro-inflammation would take a prognostic 
significance as, for example, a lower therapeutic response to minoxidil 
has been verified in subjects with male pattern alopecia [13]. Very 
important, without Biopsy, detection of peripilar signs (depression) 
through a normal Dermoscopy may be sufficient to identify these cases 
[14] (Figure 13).

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Figure 1: DLE: Koebnerization in the area of collection for autografts.

Figure 2: Pseudopelade: reactivation in the area of the grafts.

Figure 3: Lichen Planus Pilaris - reactivation in the area of the grafts.

Figure 4: Koebner phenomenon in AA: from trauma* and micro-trauma.

Figure 5: Perifollicular degranulating Mast Cells in the active phase of AA.

Figure 6: Fibrosing Frontal Alopecia – is the same patient of figure 7.

Figure 7: Fibrosing Alopecia in Pattern Distribution (FAPD): 15 years before it appeared clinically to my observation as a normal female pattern baldness.

Figure 8: FAPD- treatment with finasteride -stabilization in 6 months.

Figure 9: Alopecia Areata Androgenetica-like.
In some patients the inflammatory infiltrate has a lichenoid appearance and it is associated with fibrosis and atrophy of follicular structures, and thus assume a continuum between androgenetic forms and some lichenoid forms already mentioned [15,16,17], where androgens could still play a role, given the relative effectiveness of the 5α-reductase inhibitors - at least in the stabilization of the disease [17,18]. Interestingly, it was observed that transplantation of hair follicles from male and female human AGA on nude mice (immunologically incompetent) demonstrated a recovery of growth capacity equal or even superior to that of normal terminal hair [19]; a possible explanation of this phenomenon could be the resolution of perifollicular micro-inflammation and we therefore suggest to look more closely to inflammatory factors in triggering and maintenance of AGA. In particular an important role could be played by the fibrogenic cytokine TGFβ, proved capable under androgenic stimulation to induce catagen in susceptible follicles [20] and notoriously critical factor in healing physiological and pathological processes [21]. Moreover, an improvement of AGA is observed after Chemotherapy and is perhaps associated with the same anti-inflammatory effect of some anticancer drugs [22]. We conclude therefore that a careful dermatological evaluation of these patients, with possible help of imaging studies and any concomitant pharmacological treatments may be helpful for reducing the risk of failure or poor cosmetic results of surgical treatment of alopecia, also by means of avoiding the possibility of a KP.

References


