Telogen effluvium after contact dermatitis in the scalp

Eflúvio telógeno após dermatite de contato no couro cabeludo

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Abstract: A case of a patient with alopecia areata treated with diphencyprone, which produced contact dermatitis in the scalp, with later total regrowth is presented. After 4 months, patient presented a very intense hair loss compatible with telogen effluvium. The author calls attention for the possibility of telogen effluvium after contact dermatitis, once the production of the contact dermatitis is one of the choice treatments for alopecia areata.

Keywords: Alopecia; Hair; Dermatitis

INTRODUCTION

Telogen effluvium (TE) was described for the first time in 1961 by Kligman. It is an intense and acute hair loss, which reaches over 600 hairs per day. Usually, TE develops between three and four months after installation of the triggering agent, which may be either physical or emotional stress, medication, fever, delivery, infection, among others. In most patients, there is an abrupt stop in the anagen phase, and the hair becomes telogen, falling after a few months. Once the triggering agent is isolated, generally after two or three months, the process stops, with ensuing total recovery.

After telogen effluvium, hairs can grow synchronously, and in the future the patient might have a hair fall crisis.

Headington tries to explain the etiopathogeny of telogen effluvium considering that it may develop under five different forms: a) immediate anagen shedding; b) late anagen shedding; c) short anagen; d) immediate telogen shedding; and e) late telogen shedding.

Around 30% of patients with effluvium can present scalp pain.

In the literature, few papers have written incriminating local agents as TE triggers. The three main cited causes are: solar radiation, contact dermatitis in the scalp and hair traction.

CASE REPORT

Fifteen-year-old white male patient, born in Guarulhos (SP), who developed alopecia areata with a few sparse classic alopecia plaques in the scalp. Upon propedeutics were found: positive gentle traction test; analysis of spontaneously eliminated hairs slightly increased quantitavely, albeit with great quantity, qualitatively, of telogen hairs, many of them with Widy's sign; telogen tricogram with mace-like alteration in hair extremity; dermatoscopy revealed peladic hair, cadaverous hair and typical white fluff, resources which confirmed clinical diagnosis of alopecia areata. Treatment was made with success by means of application of topic diphencyprone at
0.025%. After intensive contact dermatitis in the scalp, including the formation of regional adenopathy, total repilation followed. After four months of treatment, when hairs were about four-centimeter long, patient present abrupt intense hair shedding, up to hundreds of hairs per day. Falling hairs were predominantly from areas that had not been affected by the previous alopecia. Initially, diffuse alopecia areata was considered; however, exams revealed telogen tricogram with a few elongated epithelial sacs, characterizing a newly-developed telogen; positive gentle traction test; elevated number of hairs in the spontaneously eliminated hairs test; upon dermatoscopy, no peladic or cadaverous hairs, or white fluff were observed; thus characterizing TE. Patient lost approximately 30% of his hairs, and after two months total repilation ensued without any type of treatment.

DISCUSSION

TE is a relatively common tricosis in the dermatology office. Usually, dermatologists pay attention to systemic factors, such as fever, drugs, medication, stress etc., and little attention is devoted to local causes. In tropical countries, ultraviolet radiation on the scalp should be remembered.\(^3,4\) A few months after scalp sunburn, patient may develop TE. Hairdos that exert traction on hairs may also trigger TE.\(^6\) Special attention should be given to contact dermatitis in the scalp. Tosti and colaborators\(^5\) described two cases of patients who present scalp contact dermatitis because of use of hair dyes. After a few months, both patients developed a picture of telogen effluvium.

Contact dermatitis in the scalp can occur for various reasons, the main ones being cosmetic manipulations, such as dyes, hair-straightening products etc. One of the choice treatments for alopecia areata is the use of local irritants, the so-called immunomodulators or immunomodulators, which produce a localized contact dermatitis, being among the main ones: antralin, nitrous mustard, dinitrochlorobenzene, squaric acid dibutylester, diphencyprone and others. In the near future, at least potentially, patient may develop TE.

In the case described here, TE developed specially in hairs that had not been affected by the previous alopecia areata. This fact seems to show that old anagen hairs are more vulnerable to TE than newly formed anagens.

In the literature, there is no description of TE occurring after immunomodulator or immunostimulant treatment for alopecia areata.

Patients suffering from alopecia areata are often treated with chemicals, the so-called immunomodulators or immunomodulators that produce contact dermatitis in the scalp. Therefore, clinician should be attentive to the possibility of telogen effluvium happening a few months after such treatment.

REFERENCES


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