

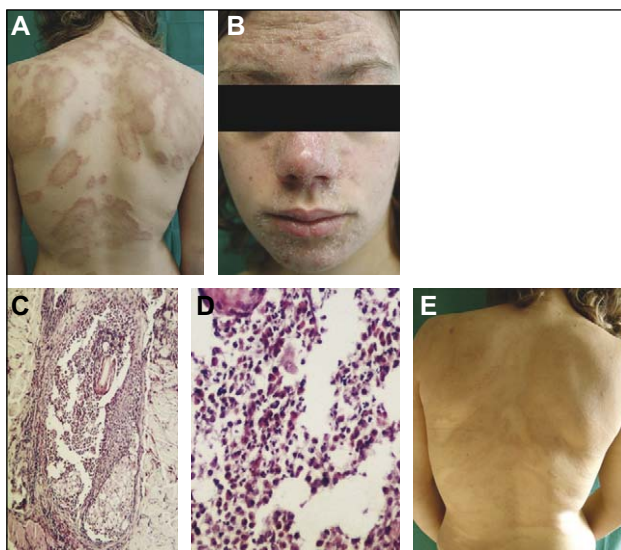


Figure 1. A) Extensive annular lesions with erythematous border. B) Papulopustular lesions on the face. C) Cutaneous biopsy showing infiltration of eosinophils around hair follicles (HE). D) Detail of eosinophilic infiltration around hair follicles. E) Excellent clinical response after 2 weeks of treatment.

cea [1]. The natural history is of chronic recurrent lesions, with occasional residual skin hyperpigmentation [3]. The pathogenesis is unclear, although some causative factors have been reported, such as infections, infestations, medications or silicone tissue injections [1, 4]. Immunosuppression associated EPF occurs with HIV infection or hematological diseases and is clinically distinct, although identical histologically [1]. Some authors claim that EPF is an inflammatory response to eliminate a yet unidentified organism, whereas others hypothesise it is essentially a Th2 cytokine imbalance [1].

An etiological treatment is unavailable, justifying the wide range of therapies with variable success [2]. For localised disease, topical corticosteroids seem adequate. With generalised disease, most of the literature favours indomethacin, 50 to 75 mg/day. As it inhibits cyclooxygenase activity, it probably prevents the formation of arachidonic acid metabolites involved in the pathogenesis of EPF [2, 5]. This drug is generally well tolerated and the response takes 1 to 2 weeks, with concomitant resolution of the eosinophilia. Therapy should be continued for up to one year with a partial response, increasing the dose to 150 mg/day or associating other therapies, such as low dose oral corticosteroids or UVB phototherapy [5, 6]. However, there may be a relapse upon indomethacin interruption, so it seems advisable to gradually taper it to the lowest possible dosage, averaging 50 mg/day [6].

We emphasise the unusual characteristics of this case, namely the patient's young age, female gender and Caucasian race, highlighting the need for a greater awareness of EPF in the western world. ■

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Systemic allergic contact dermatitis due to phenylephrine in eyedrops, with a long-lasting allergic patch test reaction

We read with interest the article by Nosbaum *et al.* [1] concerning the pathophysiology and clinical findings of allergic contact dermatitis. Allergic contact dermatitis is sometimes elicited by a topical drug, and systemic administration of a drug to which an individual has been sensitized may cause systemic allergic contact dermatitis. Here, we report the first case of systemic allergic contact dermatitis that spread over the face, neck and chest due to phenylephrine in eyedrops and was associated with a long-lasting allergic patch test reaction (LLAPTR).

The patient was a 53-year-old Japanese woman without a personal or family history of atopy who developed periorbital oedema and erythema with pruritus within 24 hours after an operation for retinal detachment. From the following day, pruritic erythema, papules and vesicles spread over the face, neck and chest. We suspected allergic contact dermatitis caused by items used perioperatively, such as antiseptics or eyedrops. Systemic treatment was not administered during the operation. Her symptoms were not responsive to treatment with topical corticosteroids, but addition of systemic corticosteroids led to a significant improvement of the symptoms after two weeks.

Patch testing was performed with the baseline series of the Japanese Contact Dermatitis Society, antiseptics, local anesthetics and Cravit[®], Tarivid[®], Flumetholon[®], Rinderon[®]-A and Mydrin[®]-P eyedrops. The results were assessed after 48 hours, 72 hours and 7 days, based on the recommendations of the International Contact Dermatitis Research Group. A positive reaction occurred with Mydrin[®]-P (as is; day 2: +++; day 3: +++; day 7: +++). To identify the precise allergens, further patch testing was carried out with ingredients provided by the manufacturer, and a positive reaction occurred with phenylephrine hydrochloride (5% pet.; day 2: +++; day 3: +++; day 7: +++). Reactivation of the initial lesions did not occur on patch testing. The positive reactions with Mydrin[®]-P and phenylephrine both persisted for two months and left residual pigmentation. The patient had previously used eyedrops containing phenylephrine, but she had not shown any symptoms. Therefore, repeated use of phenylephrine in the eyedrops may have caused the sensitization. We recommended that the patient should avoid the use of medications containing phenylephrine and there has been no recurrence of her problem.

Phenylephrine hydrochloride is a sympathomimetic drug with α -receptor stimulatory activity that is frequently used as a mydriatic and decongestant in ophthalmology. Contact dermatitis due to an ophthalmic preparation usually induces inflammation in the periocular region. There have been several reports of allergic contact blepharoconjunctivitis caused by phenylephrine [2-5], but systemic allergic contact dermatitis due to ophthalmic use of this drug has not been reported. Systemic absorption of ophthalmic solutions of phenylephrine can occur [6], and therefore the dermatitis in our case might have spread to areas other than the periocular region due to systemic exposure to phenylephrine. Thus, phenylephrine in eyedrops should be considered as a possible allergen in systemic allergic contact dermatitis. As seen in our patient, three previous reports have described LLAPTR to phenylephrine that persisted for 2 to 7 months [2-4]. The mechanism of this phenomenon is unclear. However, since contact dermatitis usually resolves after a decrease in the local concentration of the allergen and/or immunoregulatory actions by regulatory cells and cytokines [1], the LLAPTR may be associated with a defect in these mechanisms or an active sensitization effect caused by patch testing. ■

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Contact immunotherapy-induced Renbök phenomenon in a patient with alopecia areata and psoriasis vulgaris

Happle *et al.* first introduced the term “Renbök phenomenon”, derived from the reversal of “Köbner”, to describe the observation of normal hair growth in psoriatic lesions in patients with co-existing psoriasis and alopecia areata (AA) [1]. Although the exact mechanism of this unique phenomenon is unknown, it has been proposed that biological events inherent in psoriasis may act on hair follicles to restore hair growth in AA. We herein describe the Renbök phenomenon related to contact immunotherapy in a patient suffering from AA and psoriasis vulgaris.

A 15-year-old girl presented with a 3-year history of patchy hair loss on the vertex of the scalp with a gradual increase in the number of lesions. In addition, scaly and erythematous eruptions appeared on her scalp and upper extremities one year after the onset of scalp hair loss. Initial examination at our outpatient clinic revealed multiple hair loss patches on her scalp and scaly lesions on her scalp and upper extremities. Interestingly, the presence of terminal hair coincided with the scaly lesions (*figure 1A*). There was no associated systemic disease nor was there any family history of AA or psoriasis vulgaris.

Hematoxylin and eosin (H-E)-stained sections of a biopsy specimen from a hair loss patch revealed lymphocyte infiltration around atrophic hair follicles, compatible

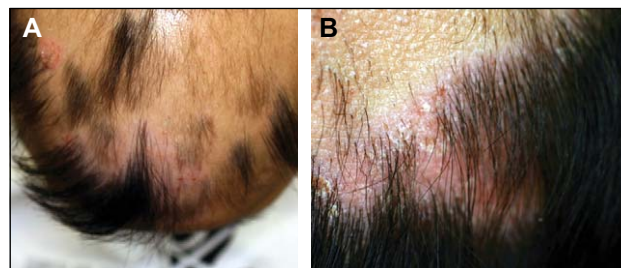


Figure 1. A) Extensive hair loss was observed on the vertex and occipital region of the head. B) Concomitant hair regrowth and psoriasis on the scalp 1 month after contact immunotherapy.