Köbner Phenomenon in a Psoriatic Patient under Cyclosporin Therapy after Contusion Trauma and Local Application of Hyperaemic Ointment

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A 39-year-old male patient had severe generalized type 1 (early-onset) psoriasis vul-garis (baseline PASI 43.9) in the acute phase of the disease; he was admitted to our department for treatment. After general and der-matological examination treatment with cyclosporin A (CyA; Sandimmune®) was started. Routine laboratory tests including creatinine clearance were normal, repeated blood pressure measurements were within the normal range. CyA therapy was given at a dose of 2.5 mg/kg/day (Sandimmune soft capsules, divided into 2 daily portions), and the patient responded well to treatment without showing adverse drug reactions. Within 12 weeks of CyA treatment psoriatic skin lesions had completely cleared (PASI 1.2).

After being on CyA treatment for 6 months the patient had an accident and hit his left chest resulting in contusion of the ribs. His physician prescribed an ointment containing nicotinate and salicylate together with flufenamic acid which the patient applied to the left side of the chest twice daily. Five days after trauma and 2 days after first use asymptomatic disseminated red papules occurred in the area where the ointment was applied.

At examination the patient was still clear of previous psoriatic lesions. The left side of the frontal chest showed disseminated single erythematous lesions of about 0.5 cm in diameter clinically resembling guttate psoriasis.

Histologi-cal investigation of a punch biopsy taken from a papule showed the typical histology of eruptive guttate psoriasis.

Routine laboratory tests including creatinine clearance were within the normal range. CyA trough blood levels were 36 ng/ml (mean; range: 78-20 ng/ml, measured by Sandimmune radio-immunoassay kit). Systemic CyA therapy was continued, and the patient received an emollient cream. Within a month the guttate lesions cleared completely.

Until stopping CyA treatment no relapse of psoriasis could be observed in the patient.

Discussion

The Köbner phenomenon is a well-known feature in psoriasis that can be elicited by physical or chemical irritation of the skin and excessive UV light exposure [for a review, see 1]. It has been emphasized already by Köbner [2] that such a reaction may occur only during the active phase of psoriasis and is rarely seen in the chronic stable phase of the disease. Guttate psoriasis following infection is also considered as a Köbner phenomenon [1, 3].

In our patient a Köbner reaction followed blunt trauma (contusion) and the application of an ointment containing nicotinate and salicylate as hyperaemic substances as well as flufenamic acid belonging to the group of non-steroidal anti-inflammatory drugs (NSAID).

Contusion triggering Köbner reactions has been mentioned by Farber et al. [4]. Although there is some debate as to whether capillary damage or dilation following trauma may lead to the appearance of psoriatic plaques [5, 6], experiments using hyperaemic substances such as nicotinate (a compound of the hyperaemic ointment used by our patient) or capsicum locally failed to induce Köbner lesions [7].

Systemic NSAID are reported to be either beneficial or worsening existing psoriatic lesions [8]. Topical application of indo-methacin caused exacerbation in 70% of the treated patients, whereas in 25% improvement of psoriatic plaques was observed [9]. For topical flufenamic acid no data concerning influences on psoriasis are available.

The development of the Köbner phenomenon in psoriatic patients under CyA therapy has, to the best of our knowledge, not been reported yet.

Our observation shows that potent stimuli such as trauma and/or chemically induced hyperaemia or topically applied NSAID may elicit the psoriatic tissue reaction despite clinically effective CyA treatment.

In fact, the pre-existing psoriatic involvement of the skin had completely cleared, whereas the occurrence of new 'Köbnerized' lesions was not prevented by continued CyA treatment. Our observation indicates that CyA therapy is at the threshold in preventing new lesions and clearing existing plaques. Consequently the trigger for the Köbner reaction observed in our patient was able to overcome CyA-induced suppression.

Since frequent relapses after CyA withdrawal are well known [10-13], this drug

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seems to clear psoriasis only while present in the body.

Our observation may also stimulate the discussion as to whether the Köbner phenomenon may represent a different type of tissue reaction which is not affected by low-dose CyA therapy.

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Letter to Dermatology