Koebner Phenomenon in Lichen sclerosus et atrophicus

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We examined a woman with lichen sclerosus et atrophicus (LSA) with manifestations in two scars. As only a few reports on the appearance of Koebner phenomenon (KP) in LSA [1,2] have been published upon yet, we should like to inform you about our case.

Fig. 1. LSA in scar after cholecystectomy.

A 55-year-old woman had an appendectomy in 1964 and a cholecystectomy in 1976. The skin affection appeared in 1985 simultaneously on the inner aspects of both wrists, then in a recent scar after an incised wound (3 months old), and in an older scar in the abdominal wall after cholecystectomy (9 years old, fig. 1). The affection consisted of whitish, flat papules, 1–4 mm in diameter, with a coarse surface, which on the wrists merged into sharply demarcated irregular lesions. At the scar sites their configuration was linear, and on the abdomen, with accessory punctated lesions in the scars after stitches as well as outside them. The papules itched, and after dissemination showed no progression. There were no genital lesions. Histological findings were typical for LSA (fig. 2).

The etiology of LSA is unknown, lesions usually appearing spontaneously without any precipitating factor. A preceding infection is supposed to play a provocative or localizing role in some cases of the genital affection. LSA has been reported in a vaccination site [1] and following trauma [2]. We have diagnosed LSA in two scars.

Scars are sites of localization to psoriasis vulgaris, lichen planus and vitiligo. Other affections are localized there less frequently: mastocytosis [3] and erythema multiforme [4]. Probably not the localization but the age of the scar is of importance. In recent scars KP is more frequent.

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Fig. 2. Histological picture with finding typical for LSA. Hema-toxylin-eosin. $\times 160$.

In our case of LSA it was positive at the sites of a 3-month-old scar after an incised wound on the dorsal aspect of the hand and of a 9-year-old scar after abdominal surgery. It was not positive in a 21-year-old scar after hypogastric surgery.

KP also occurs at recent reparation sites in cases where the character of the lesion might lead to cicatrization – cutaneous pseudolymphoma after herpes zoster [5], drug eruption after radiodermatitis [6].

A general common feature present in KP is epidermal lesion; the necessity of the presence of a dermal inflammatory reaction seems to be probable in most cases. We suppose that also local congestion from the failure of lymphatic drainage in the scar [7] may play an important role in the development of KP.

Despite its still unexplained constitution, KP remains a remarkable phenomenon, whose study may contribute to an elucidation of general pathophysiological mechanisms of the skin.

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Can C4AQO Decreased Frequency Be Considered a Genetic Marker for Vitiligo and Familial Melanoma?

Sir,

A decreased frequency of C4AQO has been found in both vitiligo and familial melanoma patients. This allele might be regarded as a genetic marker possibly acting as a resistance factor for both diseases. The serum complement proteins are an important part of the mammalian immune response being required for the lysis of antibody-targeted cells [1,2]. Some of these proteins (C2 , Bf , C4) , characterized by a high polymorphism, are encoded by genes representing the class III of the major histocompatibility complex (MHC).

Class III was reported to be involved in melanoma, with decreased Bf-FF and increased Bf-S alleles [3,4], and in familial melanoma, with a decreased C4AQO frequency [5]. Class III was referred to be involved also in sporadic vitiligo, with a decreased C4AQO frequency [6].

These observations suggest a possible genetic connection between melanoma and vitiligo , and give support to the hypothesis , already proposed by some authors [7], who stated that ‘there is a genetically determined tendency of some pigment cells to be destroyed and yield some form of vitiligo or to become transformed to melanoma cells’. These authors hypothesized an immunological process ‘by which the presence of vitiligo can counteract the proliferation of melanoma cells’. Our two works [5, 6] should give credit to this hypothesis even if many points must be clarified: the possibility to find a common clue in such different phenomena (the uncontrolled proliferation of malignant melanocytes and the destruction of melanocytes) remains a matter for speculation.

A. Among the possible explanations we consider: (1) The protein encoded by C4AQO is less efficient than those encoded by other alleles: as a result a decreased frequency of this allele leads to an above average efficiency of the immune system which would be able to contrast even if partially

malignant cells and would be responsible for an erroneous destruction of the delicate normal melanocytes. This hypothesis is confirmed by a greater survival rate of the patients with