Expression of p16 in psoriasis and chronic spongiotic dermatitis

Sir,

Several recent studies have addressed the pathogenesis of psoriasis and the interplay between keratinocyte proliferation and apoptosis. Nickoloff have shown that p16 protein is expressed in keratinocytes resistant to apoptosis, proposing that its overexpression may play a role in the development of psoriatic plaques.[1,2] $p16^{INK4a}$ gene promoter methylation was found in approximately 30% of psoriasis patients and correlated with the Psoriasis Area Severity Index.[3] The definitive role of p16 protein in psoriasis has not yet been confirmed and much of the data are inconsistent. In contrast to Nickoloff's results, Mark *et al*. found no difference in p16 gene expression in lesional and perilesional psoriatic skin through experiments with real–time polymerase chain reaction (PCR).[4] Abou EL-Ela *et al*. reported that the concentration of p16 protein measured with PCR was lower in patients with psoriasis prior to phototherapy, but was increased after treatment.[5]

Histopathological criteria for psoriasis are well-established; however, the diagnosis is challenging in certain scenarios. When considering the differential diagnosis of chronic spongiotic dermatitis, particularly in acral sites, distinguishing between these entities may be challenging. We aimed to determine if p16 protein expression is unique for the psoriasis lesions and if its expression is helpful in the differentiation of psoriasis from chronic spongiotic dermatitis in acral and nonacral sites. A retrospective search was performed, and 13 biopsies from patients with psoriasis (5 from acral sites) and 11 biopsies from patients with subacute spongiotic dermatitis (6 from acral skin) were selected. The average age of patients with psoriasis was 57.7 ± 14.3 (4 men and 9 women) and spongiotic dermatitis – 64.5 ± 15.8 years (6 men and 5 women). Periodic acid-Schiff staining was performed on all lesions to exclude fungal infection. We intended to include only patients with a primary diagnosis of psoriasis or spongiotic dermatitis and no previous treatment.

The slides were stained with the p16 antibody (clone E6H4, Roche mtm laboratories AG, Heidelberg, Germany). Overall expression of p16 protein was seen in less than 25% of the total epidermis in lesions of both psoriasis and spongiotic dermatitis. Focal expression (presence of clustered positive cells) of p16 protein was observed in 7/13 lesions of psoriasis [Figure 1] and in 3/11 lesions of spongiotic dermatitis [Figure 2] (P = 0.4, Yates corrected Chi-square, Statistica® 10, Tulsa, OK, USA).

No difference was seen in the expression of p16 between acral and nonacral lesions of psoriasis and chronic spongiotic

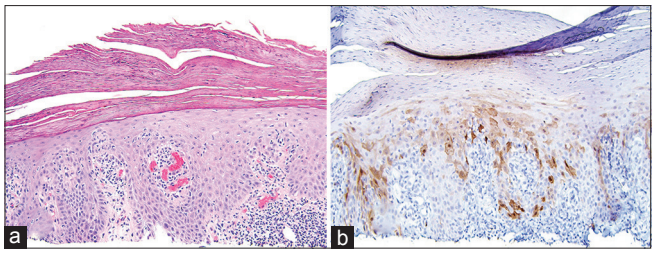

Figure 1: Psoriasis. (a) Inflammatory lymphocytic dermatitis with acanthosis. Parakeratosis and neutrophils in the stratum corneum, dilated blood vessels within the epidermis (H and E, stained slides, ×200). (b) Expression of p16 protein in psoriatic skin, p16 stained sections, ×200

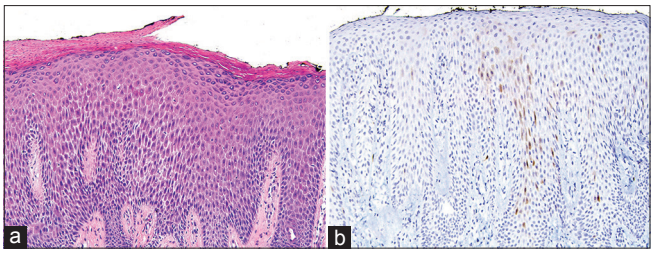

Figure 2: Spongiotic dermatitis. Inflammatory lymphocytic dermatitis with acanthosis and slight spongiosis with an overlying scale crust (H and E, stained slides, ×200). (b) Expression of p16 protein in spongiotic dermatitis, p16 stained sections, ×200

dermatitis (p16 was expressed in 3/6 lesions biopsied from acral skin of patients with spongiotic dermatitis and in 2/5 patients with psoriasis; $P = 0.8$, Yates corrected Chi-square. Review of both types of lesions revealed no differences in the pattern of p16 expression between acral and nonacral sites.

In summary, overexpression of p16 in psoriatic lesions is not a consistent finding and cannot be used in the assessment of the psoriasiform dermatitis. The presence of p16 expression in spongiotic dermatitis also suggests that it may play a role in its pathogenesis. Further investigation is necessary to evaluate the clinical significance and potential role of p16 protein in cutaneous inflammatory conditions.

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