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LETTERS TO THE EDITOR

Somatostatin Immunoreactive Cells and Merkel Cells in Psoriasis

Sir,

We have reported previously on the increase in the number of somatostatin-immunoreactive cells with dendritic morphology in psoriatic skin (1, 2). The number of Merkel cells is increased in the psoriatic epidermis and, in contrast to the situation in normal skin, a subgroup of Merkel cells expresses somatostatin (3). This prompted the question as to whether the somatostatin-positive cells in our previous reports are Merkel cells (2, 4). Neuron-specific enolase (NSE) has been reported in several studies to be a reliable marker for Merkel cells in human epidermis (5, 6). The aim of the present study was to investigate whether there is a co-localization of somatostatin and NSE among the dendritic cells in psoriatic skin, which could indicate that the somatostatin-positive cells are in fact Merkel cells.

MATERIAL AND METHODS

Punch biopsies (4 mm) were taken from lesional psoriatic skin from 6 patients with a chronic plaque psoriasis. Lidocaine without epinephrine (Astra, Södertälje, Sweden) was used as local anaesthetic. The specimens were immersed for 3 h in 4% paraformaldehyde and 14% saturated picric acid in 0.1 M Sörensen phosphate buffer (pH 7.4) at 4°C, and then rinsed in the same buffer containing 10% sucrose for at least 24 h. Sections (12 μm) were cut on a cryostat (Microm HM 500 M) and stored at −20°C. The sections were stained using an indirect immunofluorescence technique according to Coons (7). Double-labelling experiments were performed with a mixture of polyclonal rabbit antibodies against human somatostatin diluted 1:200 (Peninsula, St Helens, UK) and mouse monoclonal antibodies against neuron-specific enolase (NSE) diluted 1:25 (Dako, Glostrup, Denmark). The sections were incubated overnight with the primary antibodies in a humid atmosphere at 4°C. The sections were then rinsed and incubated for 60 min at room temperature (21°C) with a mixture of tetramethylrhodamine (TRITC)-labelled swine anti-rabbit antibodies (Dako) and fluorescein (FITC)-labelled goat anti-mouse antibodies (Dako). All antisera contained 0.3% Triton X-100. The mounting medium contained para-phenylenediamine to prevent fading of the fluorescence. The material was examined in a fluorescence microscope (Zeiss Axioplan equipped with an MC 100 camera). The experiments were repeated twice.

RESULTS

In psoriatic skin, dendritic somatostatin-positive cells were found mainly in the papillary and upper reticular dermis (Fig. 1A). A few cells were also present in the spinous and basal layers of the epidermis. NSE-immunoreactive cells were mainly seen in the basal layer of the epidermis, but also in the dermis (Fig. 1B). Except for a few weakly stained double-labelled cells in the epidermis, most of the dendritic cells did not co-express somatostatin and NSE, and no co-expression was seen in the dermis, where the majority of the somatostatin-immunoreactive cells are located (Figs. 1A and B).

DISCUSSION

The fact that the vast majority of the somatostatin-positive cells do not co-express NSE indicates that the somatostatin-positive cells and the Merkel cells represent two different groups of cells in the human skin. In previous studies we found no evidence for co-localization of somatostatin and other dendritic cell markers, such as factor XIIIa, S-100, CD1a, CD45 and CD68. Only a subgroup of the dendritic cells co-expressed somatostatin and HLA-DR (1). On the basis of these findings we conclude that the somatostatin-immunoreactive cells may represent a separate population of the dermal dendritic cells found in elevated numbers in plaque psoriasis. The function of these cells is unknown. Somatostatin has been used in several studies as treatment for psoriasis (8). Somatostatin also has several interesting immunomodulating (9) and antiproliferative (10) properties. Therefore the somatostatin-positive cells may well be of major importance in an inflammatory skin disorders with hyperproliferation, such as psoriasis.

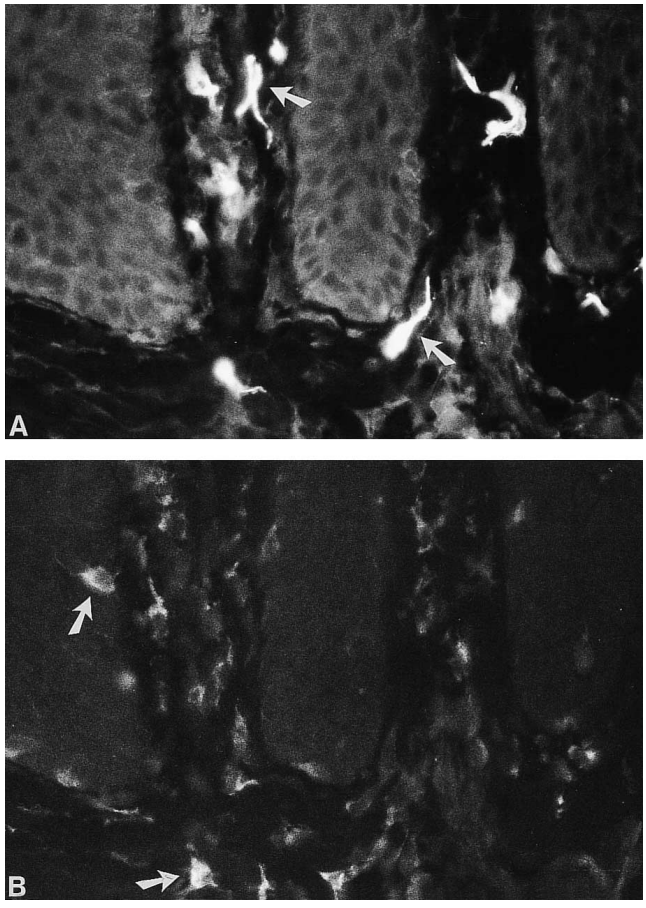

Fig 1. Immunofluorescence micrographs of a section of psoriatic skin after double-staining with antibodies against (A) somatostatin and (B) neuron-specific enolase. No coexistence can be seen. Arrows indicate some of the cells in A and B for orientation. Magnification ×400.

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Inverse Psoriasis Induced by Terbinafine

Sir,

Terbinafine is an allylamine, it is a lipophilic compound used for the treatment of onychomycoses and other fungal infections. Adverse effects are reported in 10.4% of patients, with cutaneous reactions in 2.7% (1). These include severe reactions, such as erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis (1, 2), cutaneous lupus erythematosus (3) and acute generalized exanthematous pustulosis (4). Recently terbinafine has been linked with the occurrence of psoriasis *de novo* or its exacerbation (1, 2, 5). We report here the first case of inverse psoriasis induced by terbinafine.

CASE REPORT

A 74-year-old woman, suffering from a long history of fingernail dystrophy, was treated with oral terbinafine, 250 mg/day, by her general practitioner. Onychomycosis was diagnosed clinically. No other skin lesions were noted at that time. Two weeks after the beginning of the treatment, she developed erythematous and scaling lesions of the groin, vulva, submammary folds, axillae and navel. The whole scalp showed red and scaling patches of varying size. Pitting, subungual hyperkeratosis and onycholysis of the fingernails were present. All features were consistent with a diagnosis of inverse psoriasis associated with fingernails and scalp psoriasis. There was no personal history of psoriasis, but her first cousin was affected with psoriasis. She was on no other medications. Routine blood screening was negative or within normal limits. Terbinafine was discontinued and this was followed by rapid improvement of the psoriasis which, with only topical drugs, almost completely disappeared in 2 weeks, except for the fingernail dystrophy.

CONCLUSION

In this case, a probable psoriatic onychodystrophy, misdiagnosed as onychomycosis, without mycological investigation and treated with terbinafine, induced an inverse psoriasis. We emphasize the importance of mycological investigation before commencing therapy for suspected onychomycosis. This case and other reports suggest prudence when terbinafine is used for onychomycosis or dermatophytosis in patients with coexistent psoriasis.

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