Circulating sICAM-1 during UV Therapy of Psoriasis

Sir,

Intercellular adhesion molecule 1 (ICAM-1, CD54) is considerably increased on keratinocytes and on the cellular infiltrate in the psoriatic lesion, as well as on the endothelial cells of the capillaries of the surrounding tissue (1), and decreases with clinical improvement during treatment (2). Soluble ICAM-1 (sICAM-1) in serum is increased in patients with psoriasis, but results concerning its correlation to the severity of psoriasis have been conflicting (2–5). The purpose of this study was to investigate a possible association between the PASI score and the level of sICAM-1 before and during PUVA and UVB treatment of psoriasis.

MATERIAL AND METHODS

Subjects

Twenty patients (age range 21–69 years) with chronic plaque type psoriasis were enrolled in the study after giving informed consent. Topical steroid treatment had been given before the start of the study to 13 of the patients, without success.

The study was approved by the Medical Ethics Committee of Copenhagen.

UVB and PUVA treatment

The patients were exposed to a bank of broad-spectrum UVB tubes (Philips TL 12), 3–5 times a week ($n=9$), or a bank of broad-spectrum UVA sources (Sylvania FR90T12/PUVA/HO) 1 h after ingestion of 8-methoxypsoralen 2–3 times a week ($n=11$). The UVB and the PUVA treatment was guided by skin reflectance measurements (6,7). The length of the treatment period varied from 2 to 9 weeks.

Assessment of disease severity

The severity of the disease was assessed by the PASI method (8). Each patient was scored before and weekly during the UV treatment.

sICAM-1 determination

At PASI scoring a 10-ml blood sample was collected and centrifuged within 30 min of collection. Serum was stored at −80°C and serum sICAM-1 was determined by a sandwich ELISA method (9) with minor modifications. The concentration of circulating sICAM-1 was calculated from the curve generated from optical density and the soluble standard ICAM-1 titration curve.

Statistical analysis

Correlation was tested by Spearman's test for rank correlation. Changes in sICAM-1 and PASI score during therapy were analysed by Wilcoxon's matched pairs test. The tests were considered significant when $p<0.05$.

RESULTS

Pretreatment sICAM-1 levels showed a significant correlation to pretreatment PASI score (Fig. 1). Consecutive measurements of sICAM-1 and PASI score during therapy revealed a relatively constant level of sICAM-1 throughout the study and a decrease of PASI score in both UVB and PUVA therapy. The level of sICAM-1 did not decrease significantly during treatment with either UVB or PUVA. The overall initial median level was 214.5 ng/ml (range 147–380 ng/ml) and the median level at the end of treatment was 223.5 ng/ml (range 160–340 ng/ml). The PASI score of all patients declined significantly during treatment with both UVB and PUVA. The initial and the end of treatment median PASI score was 17.5 (range 3.6–36) and 3.35 (range 0–14.4), respectively. At the end of therapy, there was no correlation between PASI score and sICAM-1.

DISCUSSION

As sICAM-1 is expected to derive from activated endothelium and inflamed tissue, it is not surprising to detect increased levels of sICAM-1 in patients with psoriasis, and the correlation of initial PASI score and sICAM-1 suggests the amount of circulating sICAM-1 to be a potential marker of disease severity. Our findings regarding the correlation between initial PASI score and sICAM-1 are in agreement with the result obtained by Schopf et al. (3). However, two other recent studies do not find any pretreatment correlation (4, 5). The lack of correlation may be explained by small ranges in pretreatment PASI scores, or the patients may have received treatment just prior to study enrolment, resulting in affected first measurement of sICAM-1. In our study the PASI score was found to decline significantly during treatment. However, the sICAM-1 level remained unaltered in our study and the studies earlier reported (3–5). The fact that sICAM-1 did not decrease, while the PASI score declined during therapy, is somewhat contrary to expectation and still needs to be explained. In vitro studies of UVB radiation of keratinocytes and endothelial cells showed initial suppression of membrane-bound ICAM-1, but after 24–48 h the expression was stimulated (10, 11). A similar in vivo stimulation by UV

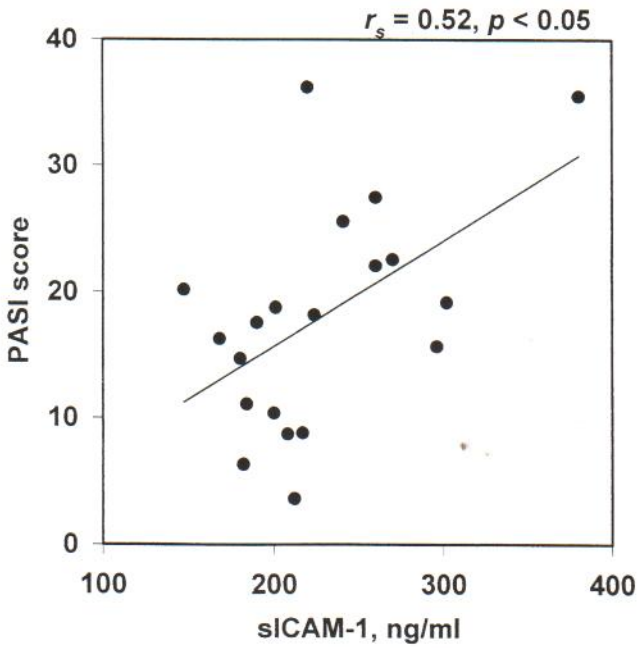

Fig. 1. Correlation between sICAM-1 and PASI score in 20 patients with psoriasis before treatment with PUVA or UVB ($r_s=0.52$, $p<0.05$).

treatment could explain a continued increased concentration of sICAM-1 in the circulation, produced by shedding of sICAM-1 from epidermal cell layers. Besides this theory, a persistently elevated level of sICAM-1, independent of successful clinical treatment, could be maintained by upregulated inflammatory mediators unaffected by therapy, like the IL2 receptor (12), and may, indeed, be a reflection of an imbalance of the cytokine network, influencing the severity of psoriasis.

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