Dear Sir,

We read with interest the letter by Yamada et al. [1]. Our experience is in contrast with the results of the Japanese colleagues. In fact, we have published in the January 1994 issue of Allergy [2] that the measurement of soluble intercellular adhesion molecule 1 (sICAM-1) levels could be a new marker, together with sIL-2R, eosinophilic cationic protein (ECP) and CD14 for monitoring atopic dermatitis (AD) [3-5]. Levels of sICAM-1 were measured by the same ELISA technique (Bender MedSystems) in sera from patients with an acute exacerbation of AD (n = 16) on admission to and discharge from our Department of Dermatology. At admission, the sICAM-1 levels in sera from patients with AD were 3.23 ± 1.32 ng/ml (range 0.35-6.15) i.e. slightly higher than those of the blood donors (n = 100) and significantly lower at discharge (p = 0.014) after improvement of the skin condition. There were no significant differences in analyzing the group of ‘pure’ AD without concomitant respiratory allergies and the group of AD with bronchial asthma [2]. On the other hand, other Japanese authors have found that circulating ICAM-1 levels gradually increase with the severity of AD and correlate significantly with ECP levels [6]. Longitudinal studies revealed a significant decrease in the levels of sICAM-1 with improvements in disease activity [7].

During a clinical trial with ciclosporin A in 13 patients with severe AD we also measured sICAM-1 levels at the beginning of treatment. The geometric mean values were also elevated, but not in all the cases (fig. 1). However, in contrast to our previous study, there was no significant drop after a course of 16 weeks of ciclosporin treatment (4-5 mg/kg/day). We believe that AD is a hetero-genous atopic disease with many subtypes, both extrinsic and intrinsic [8], with or without defect in the linoleic acid metabolism [9] and, therefore, with different cytokines or mediator release according to the different subtypes [8]. Further studies in a greater number of AD patients are needed to better characterize the different subtypes with respect to their individual mediator and cyto-kine profiles.

Fig. 1. Levels of sICAM-1 in the serum of patients before and after 16 weeks of ciclosporin A treatment. The sICAM-1 levels were initially increased (O) (mean: 262.9± 51.5 ng/ml, n = 13) and decreased after 16 weeks of ciclosporin A therapy (∗) (mean: 242.7 ± 40.3 ng/ml), n = 13). The difference, however, was not significant.
References

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