Epidermal Growth Factor Concentration in the Sera of Male Psoriatic Patients

Sir,

Epidermal growth factor (EGF) is found in various organs, including skin, and also in body fluids, such as blood. The main EGF reservoir are platelets (granules), from where it is secreted, together with other growth factors (PDGF, $TGF-\beta$) (1).

The concentration of EGF in blood serum of male psoriatic patients was compared with the concentration in a control group, to establish the correlation and the PASI score. The studied group of men with psoriasis was divided into two subgroups according to relapse duration—less than 2 months, versus more than 3 months.

MATERIAL AND METHODS

Male patients were chosen in order to eliminate possible hormonal influence. The study was conducted on 46 patients with a mean PASI score of 29–30 and 20 healthy males of the same age.

The concentration of EGF in blood serum was evaluated by the radio-immunoassay procedure in duplicates with the use of J 125 hr EGF Reagent Pack for RIA (Amersham, code IM 1961/, sensitivity threshold 0.08 ng/ml). The EGF standards–10.0 ng, 5.0 ng, 2.5 ng, 1.25 ng, 0.6 ng, 0.3 ng, 0.16 ng and 0.08 ng-were produced with the use of human lyophilized EGF (EGF Human Recombinant, Sigma, code E-1264), diluted with Eagle MEM 1965 medium. The activity of samples labelled with J 125 was measured with γ-radiation counter, Gamma Automat NRG 603 Tesla. Statistica programme was employed to perform statistical analysis. For statistical analysis, Student *t*-test and *r* Pearson's correlation coefficient were carried out.

RESULTS

In the studied group no statistically significant differences between EGF concentration in acute and chronic psoriatic patients and healthy volunteers were observed $(0.19\pm0.01~\text{ng/ml})~\text{vs}~0.18\pm0.01~\text{ng/ml})$ (mean \pm SEM). The analysis of the correlation between EGF concentration and the extension of psoriatic process expressed with the PASI score showed no dependence between these parameters. Equally, no correlation was observed between EGF concentration and relapse duration, or between EGF concentration and age of the controls, or between EGF concentration and age of psoriatic patients.

DISCUSSION

In the latest literature, numerous studies deal with the role of EGF in the pathogenesis of psoriasis. In their recent works Kulke et al. (2) strongly support the hypothesis that increased or aberrant activation of the EGF receptor pathway is sufficient for the development of epidermal hyperplasia and may contribute to similar changes observed in inflammatory skin diseases.

Earlier, when studying the expression of the EGF receptor in psoriatic epidermis, we found an almost double increase of the number of EGF receptors in adjacent "normal-appearing" epidermis, compared to epidermis with active plagues. In "normal-appearing" epidermis we also observed a significant increase of tyrosine kinase concentration stimulated by EGF,

Table I. Characteristics of studied subjects

Group	n	Age range	Age (years) M±S D	Mean PASI M±S D	Percent of body surface (lesions)	Mean relapse duration in months
Control	20	19.0-	31.4	0	0	0
C		50.0	± 9.2			
Psoriasis	46	17.0 -	35.3	29.2 40.6 ± 21	40.6 ± 21.9	9 3.62 ± 6.04
total PT		51.0	±8.9	± 9.8		
Acute	20.000.000	17.0-	35.9	28.8	39.0 ± 24.2	1.17 ± 0.63
psoriasis AC		50.0	± 8.6	±11.0		
Chronic	soriasis	17.0-	34.3	30.0	43.1 ± 18.4	6.33 ± 7.71
psoriasis CP		51.0	±9.6	±7.61		

PASI-Psoriasis Activity and Severity Index.

which is an indicator of the EGF receptor activity (3). The question we posed was if the increased number of EGF receptors or/and their increased activity in epidermis affected EGF concentration in blood serum.

With the use of a radioimmunoassay procedure, Dailey et al. (4) studied EGF in the circadian urine of patients with psoriasis, in whom changes of EGFr concentration in urine were found. The changes of EGF concentration did not correlate with the clinical condition of patients.

Venier et al. (5) treated 20 patients with grave forms of psoriasis with somatostatin and achieved a significant improvement of their condition. They carried out circadian measurements of EGF concentration in blood serum before, during and after the treatment. The EGF concentrations were normal before the treatment, whereas they significantly decreased after the treatment.

Our results confirm the observations by Venier et al. (5), who stated that EGF concentrations did not differ from normal ones. It is interesting to note the decrease of EGF serum concentration after the treatment. The EGF concentrations observed in the sera of our patients before the treatment also did not significantly differ from the controls, although they were somewhat increased. In the light of the obtained results, we can presume that in psoriasis, despite the excessive proliferation in epidermis and abnormal localization of EGF receptors, no translocation of EGF to blood serum occurs-regardless of the extension and intensity of the psoriatic lesions—but paracrine-autocrine secretion takes place (5).

REFERENCES

- Oka T, Orth DN. Human plasma EGF/beta-urogastrone associated with blood platelets. J Clin Invest 1983; 72: 249–259.
- Kulke R, Bartels J, Schluter C, Christophers E. Overexpression of the EGF-receptor cDNA in foreskin derived keratinocytes causes epidermal hyperplasia in raft culture. Book of abstracts. 26th Annual Meeting of European Society for Dermatological Research Amsterdam 1996; 161: 85.
- 3. Miturski R, Pietrzak A, Krasowska D, Lecewicz-Toruń B. The

presence of epidermal growth factor receptors (EGF-r) and tyrosine

kinase activity stimulated by EGF(EGF-TK) in psoriatic epidermis. Preliminary findings. J Eur Acad Derm Venereol 1996;7, Suppl. 2: 191.
4. Dailey GE, Kraus JW, Orth DN, Homologous radioimmunoassay

Endocrinol Metab 1978; 46: 929–936.
Venier A, De Simone C, Forni A, Ghirlanda G, Uccioli L, Serri F, et al. Treatment of severe psoriasis with somatostatin: four years of experience. J Arch Dermatol Res 1988: 280 [Suppl]:S 51–54.

for human epidermal growth factor (urogastrone). J Clin

Accepted May 16, 1997.

Poland.

Aldona Pietrzak¹, Barbara Lecewicz-Toruń¹, Grażyna Chodorowska¹ and Roman Miturski²

¹Clinic of Dermatology and ²Second Clinic of Gynecological Surgery, Medical School, Lublin, 13 Radziwiłowska Str., 20–080 Lublin,