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In Vivo Mobilization of Polymorphonuclear Leukocytes in Psoriasis: Relationship to Clinical Parameters and Serum Inhibitory Factors

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Mean migration of polymorphonuclear leukocytes (PMNL) toward autologous and homologous control sera, evaluated by quantitative skin window chamber technique, was only slightly reduced in 60 patients with psoriasis as compared to 27 normal controls ($p < 0.1$). A significant decrease in cell migration was found (1) in patients with actively spreading lesions, (2) in patients with extensive lesions involving more than 40–60% of the skin surface, (3) in the first 2 months of relapse, and (4) 5–6 months after onset of new lesions. However, PMNL migration was increased when psoriatic lesions lasted 3–4 months. Seventy-one percent of psoriatic sera exerted a suppressive effect on the psoriatic and normal PMNL migration. The inhibitors were found predominantly in patients with stationary and long-standing lesions. Some of the psoriatic sera had a stimulatory effect on the chemotaxis of psoriatic PMNL. These sera originated from those patients with active spreading lesions in the first 2 months of relapse.

These data indicate that neutrophil migration is abnormal in the course of psoriasis and that it could be modified by different proportions of both inhibitors and stimulators of chemotaxis.

Polymorphonuclear leukocytes (PMNL) seem to play an important role in the pathogenesis of psoriasis. They are a constant constituent of infiltrates of early psoriatic papules [1,2]. Their chemotaxis in vitro toward different attractants [3–6], their adherence [7], oxidative system function [8], and the intracellular content of neutral proteinases [9] were found to be increased.

In in vitro chemotaxis studies using the Boyden chamber method, inhibitors and stimulators of PMNL migration were detected in psoriatic serum [10–13]. Stimulatory activity was directed toward psoriatic neutrophils [12] and also toward normal cells [13]. The inhibitors of PMNL function were present in patients with widespread [11] and long-standing [13] psoriatic lesions.

Breathnach et al [14], using a quantitative skin window chamber technique, have found that in vivo migration of PMNL toward 50% autologous serum in patients with psoriatic lesions involving more than 25% of the skin surface was lower than in patients with minor skin involvement and in normals. In contrast, in cases of untreated psoriasis Dubertret et al [15] reported on an increased penetration of cells within 8 h through skin window. More detailed studies on larger series of patients with an account of clinical parameters have not been reported.

The purpose of our study was to analyze by the quantitative skin window chamber technique: (1) migration of PMNL as related to the activity of the disease, the duration of the relapse, and the extent of psoriatic lesions, and (2) the effect of normal and psoriatic sera on PMNL migration through the skin window.

MATERIALS AND METHODS

Selection of Patients

Studies were performed in 80 patients with psoriasis vulgaris and in 49 healthy volunteers. The patients had not received systemic medication for at least 3 months prior to the experiment. Topical treatment with corticosteroids, salicylic acid, anthralin, and tar was withdrawn 2 weeks prior to testing. The clinical state of the patients was evaluated according to the activity of the disease, as well as the extent and onset of the last relapse. Activity was determined by 2 observers and graded as: A2, mainly widely disseminated papules and pinpoint lesions; A1, peripherally spreading large psoriatic plaques, occasionally with small papules in the surrounding area; and A0, stationary skin lesions. The extent of cutaneous involvement was defined as: less than 10%, 11–20%, 21–40%, 41–60%, and more than 60%.

Quantitative Skin Window Chamber Technique

A modification of the method described by Mass et al [16] was applied. Approximately 500 experiments were done, 3–6 on each patient. The volar surface of the forearm was moistened with water for 1 h to facilitate tape stripping. The skin was treated with Betadine and 70% ethyl alcohol. Stripping was performed over a template with prepared square holes (1 cm^2), until capillary loops became visible through the glistening surface of the skin. The test sites were next swabbed with saline to remove fragments of the epidermis. Sterile glass chambers of our own design were fastened with tissue glue over the test sites and filled with media to 2 cc total volume. After 8 h, fluid in the chambers was aspirated and suspended cells were counted in a hemocytometer. Fluid, after 8 h, was found to be sterile. Cell smears prepared from each test were stained by the May-Grünwald-Giemsa method and also stained for peroxidase activity. Differential cell counts of leukocytes migrating during the 8 h showed that more than 93% of the cells were PMNL, as reported previously [17].

Results were presented as the total number of leukocytes migrated per 1 cm^2 skin window within 8 h in each chamber. The reproducibility of the technique was satisfactory.

Chemoattractive Media

A variety of media, including 50% autologous serum (psoriatic or normal), 50% pooled homologous serum from normal donors, 50% psoriatic homologous serum, and 0.5% casein (Sigma Chemical Corp., St. Louis, Missouri) in Hanks' balanced salt solution (HBSS) were used. Sera were stored in portions at −40°C; before the experiment they were diluted with an equal volume of HBSS. Some sera were heat-inactivated.

RESULTS

Migration of PMNL Toward Various Media in Psoriatic Patients and Normal Volunteers

In 60 patients with psoriasis, mean neutrophil mobilization through skin window by 50% autologous serum (5.03 × 10^6 PMNL/cm^2) and by 50% normal homologous serum (4.91 × 10^6 PMNL/cm^2) was only slightly reduced in comparison with normal volunteers (6.06 and 5.72 × 10^6 PMNL/cm^2, respectively). An approximately 2-fold lower migration of cells was found when casein was used instead of serum, both in psoriatics (2.45 × 10^6 PMNL/cm^2) and in normals (2.41 × 10^6 PMNL/cm^2). Only a few migrating PMNL were present in the medium alone (Table I).

In patients with psoriasis, there was a direct correlation between neutrophil mobilization by casein and by 50% sera—both autologous and normal. The higher the cell migration
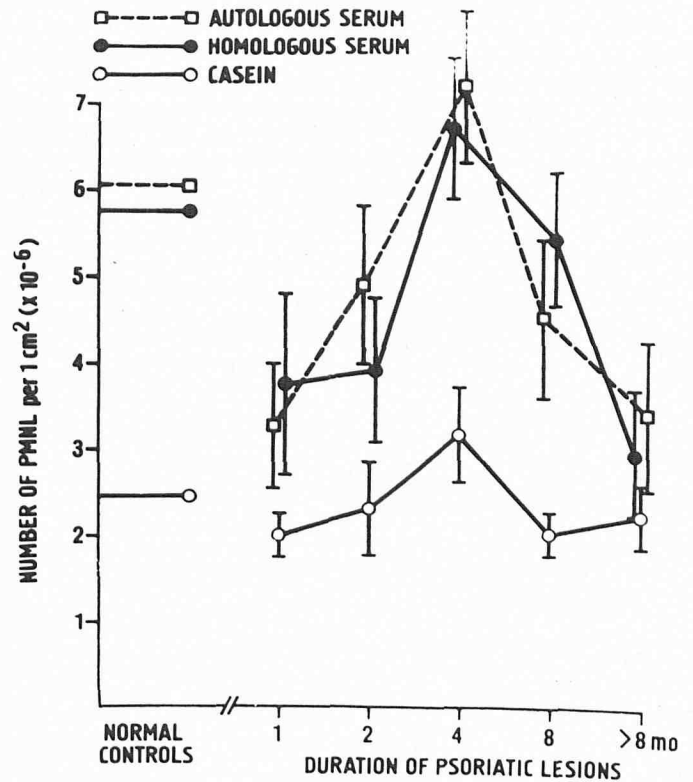

FIG 1. Number of PMNL penetrating through the skin window as related to the onset of the last relapse (mean ± SEM). For the autologous serum (□- - - -□), values were statistically different at <1 month and >8 mo vs. 2–4 months ($p < 0.02$) and vs. normal controls ($p < 0.05$); for the homologous serum (●——●), >8 months vs. 2–4 months and/or normal controls ($p < 0.02$); and for casein (○——○), 2–4 months vs. 1–2 months ($p < 0.05$).

through the skin window to casein, the greater the migration to sera.

Relationship of PMNL Chemotaxis to Duration, Extent, and Activity of Psoriatic Lesions

The number of PMNL in chamber media in patients with psoriasis was analyzed with respect to the duration of recent relapse (Fig 1). In the first 2 months after the appearance of psoriatic lesions, the migration of neutrophils to autologous and homologous sera was significantly lower in comparison with that of the control group. In contrast, an increased number of cells was found in patients with a relapse period of about 4 months duration. However, if the relapse lasted longer than 5–6 months, autologous and homologous sera induced much lower cell migration through the skin window to the chamber than in normals.

Similar, although lower, peak increases of PMNL chemotaxis about 4 months after the onset of lesions was found in experiments with casein (Fig 1).

Patients with involvement of more than 60% of the total skin surface were shown to have a 2-fold reduction of neutrophil migration toward the autologous serum (2.44 × 10^6 PMNL/cm^2) through the skin window as compared with normal controls. Significantly reduced PMNL chemotaxis toward normal homologous serum was present also in the patients with more than 40% of the skin involved. The remaining patients had cell numbers within the normal range (Table II).

In active psoriasis (A2), migration of PMNL toward the homologous serum (5.62 × 10^6 PMNL/cm^2) was similar to that of normal PMNL attracted by the normal serum (5.72 × 10^6 PMNL/cm^2). Response to casein in this group did not differ from the migration in normal controls (2.18 and 2.41 × 10^6 PMNL/cm^2, respectively). In contrast, chemotaxis of psoriatic

TABLE I. *Migration of PMNL through skin window in patients with psoriasis and in normal controls*

| Fluid in chamber | Number of PMNL × 10^{-6} per 1 cm^2 | | | | t-test |
| | Psoriasis (60) | | Normals (27) | | |
	Mean	SD	Mean	SD	
Medium	0.17	0.09	0.21	0.11	NS
Autologous serum	5.03	3.39	6.06	3.09	NS
Homologous serum	4.91	3.23	5.72	2.71	NS
Casein	2.45	1.72	2.41	1.39	NS

TABLE II. *Number of PMNL migrating through skin window as related to the extent of psoriatic lesions (n = 60)*

Fluid in chamber	Number of PMNL $\times 10^{-6}$ per 1 cm^2 (mean ± SD)					
	Extent of psoriatic skin lesions					Normals (27)
	10% (11)	11–20% (12)	21–40% (20)	41–60% (12)	>60% (5)	
Autologous serum	5.84 ± 2.88	5.55 ± 3.28	4.94 ± 3.57	4.40 ± 3.94	2.44 ± 1.84[a]	6.06 ± 3.09
Normal homologous serum	4.98 ± 3.40	4.76 ± 2.60	6.13 ± 3.29	2.98 ± 2.19[b]	1.92 ± 1.78[a]	5.72 ± 2.71
Casein	2.64 ± 1.76	2.01 ± 0.44	2.88 ± 2.09	1.70 ± 1.49	2.05 ± 0.80	2.41 ± 1.39

[a] Statistical comparison (*t*-test), mean migration different from normal controls: $p < 0.02$.
[b] Statistical comparison (*t*-test), mean migration different from normal controls: $p < 0.01$.

TABLE III. *Migration of PMNL through skin window in patients with psoriasis related to the activity of the disease (n = 60)*

Fluid in chamber	Number of PMNL $\times 10^{-6}$ per 1 cm^2 (mean ± SD)			
	Activity of psoriatic lesions			Normals (27)
	0 (12)	1 (30)	2 (18)	
Autologous serum	4.82 ± 2.25	5.50 ± 3.54	2.94 ± 2.90[a]	6.06 ± 3.09
Normal homologous serum	4.14 ± 3.15	5.01 ± 3.13	5.62 ± 3.89	5.72 ± 2.71
Casein	1.72 ± 0.54	2.59 ± 1.89	2.18 ± 1.15	2.41 ± 1.39

[a] Statistical difference A2 vs. normals, $p < 0.02$.

TABLE IV. *Inhibitory effect of homologous psoriatic sera on psoriatic and normal PMNL migration through skin window*

Initial PMNL chemotaxis[a] (no. of cells per 1 cm^2)	Group (no. of cases)	Number of PMNL $\times 10^{-6}$ per 1 cm^2 (mean ± SD)		
		Autologous	Normal homologous	Psoriatic homologous[b]
<6 × 10^6	Psoriasis (29)	2.68 ± 1.29	3.74 ± 2.63	3.83 ± 3.05 (55)
	Normals (12)	3.04 ± 1.91	3.31 ± 2.32	3.47 ± 2.37 (28)
>6 × 10^6	Psoriasis (21)	8.71 ± 1.94	7.56 ± 3.17	5.55 ± 2.76 (45)[c]
	Normals (16)	8.43 ± 1.78	6.77 ± 2.28	4.74 ± 2.30 (38)[d]

[a] Psoriatic sera were tested in the subjects with high and low initial PMNL migration.
[b] In parentheses, number of sera tested.
[c] Statistical significance (*t*-test): psoriatic vs. autologous serum, $p < 0.001$; psoriatic vs. normal homologous serum, $p < 0.002$.
[d] Statistical significance (*t*-test): psoriatic vs. autologous serum, $p < 0.001$; psoriatic vs. normal homologous serum, $p < 0.001$.

TABLE V. *Effect of homologous psoriatic serum on PMNL chemotaxis in patients with psoriasis as related to disease activity and extent of psoriatic serum donors*

Disease parameters in donors of psoriatic homologous serum	No. of cases	Number of psoriatic PMNL $\times 10^{-6}$ per 1 cm^2 (mean ± SD)[a]		Percent of patients with increase of PMNL chemotaxis[b]
		Autologous serum	Psoriatic homologous serum	
Activity of disease				
A2	14	7.36 ± 3.11	7.73 ± 3.67	57%
A1	26	8.73 ± 1.89	6.02 ± 2.95[c]	15%
A0	11	8.31 ± 2.43	3.94 ± 1.48[c]	18%
Duration of disease				
<2 months	19	7.67 ± 3.01	7.26 ± 3.22	53%
2–4 months	17	8.64 ± 1.87	5.95 ± 2.72[d]	12%
>4 months	15	8.59 ± 1.99	4.59 ± 1.22[c]	13%

[a] Psoriatic sera were tested in psoriatics with high initial PMNL migration.
[b] Above autologous serum-induced migration.
[c] Statistical comparison (*t*-test): psoriatic vs. autologous serum, $p < 0.001$.
[d] Statistical comparison (*t*-test): psoriatic vs. autologous serum, $p < 0.005$.

neutrophils toward autologous serum (2.94 × 10^6 PMNL/cm^2) was significantly decreased (Table III).

In patients with stationary lesions (AO), there was a reduction in psoriatic PMNL counts, both in autologous (4.82 × 10^6 PMNL/cm^2) and normal homologous (4.14 × 10^6 PMNL/cm^2) sera as well as in casein (1.72 × 10^6 PMNL/cm^2).

Effect of Homologous Psoriatic Sera on Psoriatic PMNL Migration

The PMNL chemotaxis through skin window toward 100 homologous psoriatic sera was tested in 50 patients with psoriasis and 28 normal volunteers. The results were compared with the PMNL migration toward autologous serum and normal homologous serum on each individual studied (Table IV).

Because of the wide range of results, patients were divided into 2 groups on the basis of an initial PMNL migration to autologous serum—lower than 6 × 10^6 cells per 1 cm^2 and higher than 6 × 10^6 cells per 1 cm^2. The number of PMNL was significantly reduced in patients with psoriasis in the presence of homologous psoriatic sera (5.55 × 10^6 PMNL/cm^2) in comparison with the migration to autologous serum (8.71 × 10^6 PMNL/cm^2) as well as to normal serum (7.56 × 10^6 PMNL/cm^2), if the subjects tested revealed a high initial PMNL chemotaxis. When the initial PMNL migration to autologous serum did not exceed 6 × 10^6 cells per 1 cm^2, the migratory cell counts were not significantly different in all sera tested: autologous, normal homologous, and psoriatic homologous.

Psoriatic sera induced much lower migration (4.74 × 10^6 PMNL/cm^2) in normal controls than either autologous (8.43 × 10^6 PMNL/cm^2) or normal homologous sera (6.77 × 10^6 PMNL/cm^2).

This psoriatic serum-dependent decrease in the migration of psoriatic PMNL of unchanged initial motility was analyzed with respect to clinical parameters of psoriatic serum donors (Table V). The reduction in cell count induced by psoriatic sera as compared to autologous serum-induced chemotaxis was found when the sera were obtained from patients with stationary (A0—3.94 × 10^6 PMNL/cm^2) and peripherally spreading (A1—6.02 × 10^6 PMNL/cm^2) lesions, as well as in patients with psoriatic lesions of more than 2 months duration (5.95 × 10^6 PMNL/cm^2).

Numerous sera of patients with actively spreading lesions (A2-57%) and patients with a recent relapse not exceeding 2 months (53%) stimulated the PMNL migration in comparison with the initial migration due to autologous serum. However, the mean PMNL counts were within normal range in these patients (Table V).

There was no relationship between the decrease in the PMNL migration in the presence of psoriatic sera and the extent of psoriatic lesions of serum donors.

When psoriatic sera were tested in normal subjects by the skin window technique, the reduction of PMNL chemotaxis was present in all subgoups of patients, irrespective of the activity of disease and/or extent and duration of recent relapse of psoriatic lesions (data not shown).

DISCUSSION

No significant difference was found between patients with psoriasis and the normal population in in vivo PMNL migration toward autologous and normal homologous serum. This is in agreement with the data of Breathnach et al [14], who found the mean PMNL chemotaxis to be normal in 50 psoriatics using the same quantitative skin window chamber technique.

However, there was a markedly decreased mobilization of PMNL in 64% of patients, while 31% of cases had increased

mobilization. These disparate reactions occurred during recent relapse of psoriatic lesions. The PMNL migration was diminished in the first 2 months of relapse, elevated at 2.5–4 months, and finally markedly depressed after 4 months. It would thus appear that selection of patients is a possible explanation for the increased PMNL migration through skin window reported in 9 psoriatic patients by Dubertret et al [15].

The time parameter was also found to be important for the other correlations observed: the PMNL migration vs. the extent as well as the activity of psoriatic lesions. A majority of the patients with long-lasting skin lesions and low PMNL migration usually had more than 40–60% of the skin involved and an inactive disease (AO). The patients with low PMNL migration, in whom the relapse period did not exceed 2 months, were found to have predominantly actively spreading lesions (A2).

The decreased mobilization of psoriatic PMNL toward serum in psoriasis could be dependent upon: (1) in vivo depression of random PMNL motility; (2) increased content of serum inhibitors of chemotaxis in psoriatic serum; (3) lowered chemoattractive activity of psoriatic serum; (4) excessive stimulation of circulating PMNL by one chemoattractant in vivo, which made the cells unresponsive to the other stimuli such as fresh human serum; (5) increased activity of tissue inhibitors of PMNL, etc.

Random PMNL motility seemed to be unchanged in the patients with active psoriasis (A2) and in those with a relapse lasting less than 2 months as determined by the response of PMNL to normal homologous serum and/or casein. However, either the reduced activity of chemoattractants in psoriatic sera or the increased amount of inhibitors of chemotaxis could be responsible for migration of PMNL toward psoriatic serum to be less than toward normal serum in the patients with the most active lesions (A2). A depletion of some chemotactic components of complement might be suggested on the basis of the data of Goldberg et al [17], who found that about 90% of chemotactic properties of human sera is dependent on the C5a activity.

The elevated chemotaxis of PMNL between 2 and 4 months of the relapse seemed to be due to the neutrophil hyperreactivity to chemotactic stimuli, as described previously [6,10–12]. Generation of prostaglandins such as leukotriene B4 [18], and neutral proteinase-produced chemotactic factor [19] could be responsible for the stimulation of PMNL in some periods of disease as well as for the accumulation of PMNL in the psoriatic epidermis.

In long-lasting psoriasis of about 6 months duration, the PMNL migration was lowered irrespective of the serum used in the chamber. This may be a result of in vivo decreased activity of cells at least in part related to inhibitors in psoriatic sera. Those inhibitors could be identical to the inhibitory factor associated with widespread psoriatic lesions, which was demonstrated by the in vitro Boyden chamber method [11,13]. The inhibition of PMNL migration might be related also to the increased serum IgA, which was found to suppress in vitro chemotaxis [13].

The presence of inhibitors of chemotaxis in the psoriatic sera was documented in acute plaque (A1) and in stationary (A0) psoriasis as well as in patients in whom the relapse lasted more than 2 months. The inhibitory factors of psoriatic sera could depress the PMNL migration only in the patients with psoriasis and/or normal controls who had normal initial migration of PMNL toward autologous or normal serum.

Our data also give some evidence for the presence of stimulatory factors in the psoriatic sera of patients with guttate psoriasis (A2) whose psoriatic lesions do not exceed 2 months duration. These factors were directed only to psoriatic PMNL, not to normal cells (data not shown). The activators of chemotaxis were previously described in psoriasis by Kawohl et al [12] which were active toward psoriatic [12] or normal [13] PMNL.

The mechanism of the decreased or increased PMNL migration in different forms and periods of psoriasis and the relationship of PMNL migration to cell activation and serum factors remain to be elucidated.

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