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The Coexistence of Psoriasis with Lupus Erythematosus and Other Photosensitive Disorders

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Psoriasis affects approximately 0.1% to 3% of the general population and has a mild geographic variation (1, 2); it is usually photoresponsive, improving with phototherapy. A small population of patients have idiopathic photosensitive psoriasis or psoriasis associated with photosensitivity of some coexistent identifiable cause. In 1927, O'Leary (3) described one of the first cases of coexistent psoriasis and lupus erythematosus (LE). Similar cases have been reported since then (4–29). Likewise, psoriasis has been documented to coexist with other photosensitive disorders (30), including vitiligo (31–37), porphyria (38–41), and drug and photocontact reactions (42–57). The coincidental occurrence of conditions such as "fair skin type" (58), polymorphous light eruption (PMLE), chronic actinic dermatitis, solar urticaria, and actinic prurigo should also be considered in patients with psoriasis who have apparent sun sensitivity.

Estimates of the prevalence of photosensitivity among patients with psoriasis have varied from 14% to 24% in older studies (59–63). These estimates have been generated primarily from studies investigating multiple aspects of psoriasis without specifically emphasizing photosensitivity or attempting to define underlying causes. Other early reports (64, 65) of photosensitive psoriasis have focused on the photodistributed localization of psoriasis and the significance of fair-skin types.

In the most thorough recent characterization of photosensitive psoriasis to date, Ros et al. (66–69) defined photosensitive psoriasis as "psoriasis in which the lesions deteriorate after sun exposure or new lesions appear." Photoirritable psoriasis (that is, pustular and erythrodermic variants) and all concomitant photodermatoses must be excluded. Using this definition, they estimated that the prevalence of photosensitivity among patients with psoriasis was 5.5% among 1,760 respondents to a questionnaire and interview. They postulated that the higher prevalence in previous studies was due to concomitant photodermatoses. They found a statistically significant increase in the photosensitive group of type I skin, psoriasis affecting the hands, heredity for photosensitivity, and advanced age. Among their photosensitive patients, 43% had a history of PMLE with secondary exacerbation of psoriasis (67).

The purpose of the present study was to examine the clinical aspects and significance of the coexistence of photosensitive disorders, including LE, in a large cohort of patients with psoriasis.

METHODS

A computerized review was performed of all dismissal diagnoses of psoriasis among patients seen at the Mayo Clinic in Rochester, Minnesota, between 1979 and 1989. Each chart of patients who had a diagnosis of photosensitivity of any type or LE was then reviewed. This review included dermatologic, general medical, and laboratory evaluations from the time of diagnosis until the most recent clinic visit.

RESULTS

During the 10-year study, 9,420 patients with psoriasis were seen, and 65 (0.69%) of these had photosensitive disorders, including LE (Table I). Of these, 23 (35%) had psoriasis and non-lupus-related photosensitivity. Forty-two (65%) had psoriasis and LE, with or without photosensitivity. The mean duration of follow-up was 15 years (range, 0 to 52 years, including patients seen for single evaluation only and those initially seen before and also after 1979). Ninety-five percent of patients were white.

Characteristics of psoriasis

The characteristics of psoriasis in this group of patients are summarized in Table II. There was a strong predominance of plaque-type psoriasis, and the mean age at onset for patients with this type was 40 years. The only significant difference in the age at onset of plaque-type psoriasis occurred in patients with systemic LE, in whom the mean age at onset of psoriasis was 33 years among 11 female patients and 54 years among 6 male patients (data are not shown in Table II).

Psoriasis was mild (less than 5% body surface area affected [BSA]) in 28 (43%) of the 65 patients, moderate (5% to 15% BSA) in 17 (26%), and severe (more than 15% BSA) in 20 (31%). A positive family history of psoriasis was found in 29% of patients, the family history was negative in 42%, and family history was not mentioned in 29%.

The location of plaques at the onset of psoriasis and subsequently is indicated in Table III. Of the 56 patients with initial plaque-type psoriasis, 38 had subsequent plaque-type disease only, 7 experienced at least one episode of erythrodermic psoriasis, 3 experienced some subsequent form of pustular psoriasis, 1 had development of inverse psoriasis, and 7 patients had no follow-up. Patients with other forms of psoriasis tended to maintain the same form for the duration of their disease.

In 34 patients, at least one biopsy specimen was taken of

Table I. *Sex and age distributions of 65 patients with psoriasis and photosensitivity or lupus erythematosus*

			Age at onset, year	
Sex	No.	%	Range	Mean
Male	24	37	13–79	44
Female	41	63	5–77	40
Total	65		5–79	41

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Table II. *Characteristics of psoriasis in 65 patients with photosensitivity or lupus erythematosus*

Type at onset	No. of patients	%	Sex		Age at onset		
			Male	Female	Range		Mean
Plaque	56	86	23 (41%)	33 (59%)	5–79		40
Generalized pustular	1	1.5	...	1		41	
Localized pustular	3	5	...	3	50–77		61
Guttate	3	5	...	3	6–36		19
Inverse	1	1.5	1	...		67	
Palmar/plantar	1	1.5	...	1		69	
Total	65		24	41			

Table III. *Location of plaques at onset of psoriasis and subseqeuntly in 56 patients*

Location of plaques[a]	Involvement at onset ($n=56$)		Involvement subsequently[b] ($n=43$)	
	No.	%	No.	%
Extremities	42	75	39	91
Scalp	27	48	28	65
Trunk	14	25	26	60
Head/neck (excluding scalp)	4	7	4	9
Intertriginous	1	2	5	12
Other[c]	6	11	7	16

[a] Initial sites were not documented in two cases.
[b] Patients with primarily plaque-type psoriasis subsequently.
[c] Groin, intergluteal cleft, umbilicus, hands.

an area of suspected psoriasis: 28 (82%) had at least one specimen diagnostic of psoriasis, 3 had results that were interpreted as "suggestive of psoriasis," and 3 had non-diagnostic results.

Photosensitivity

We defined photosensitivity as any adverse cutaneous eruption due to exposure to ultraviolet light (UVL). A history of photosensitivity was found in 44 of the 65 patients: 21 with LE and 23 with no evidence of LE. These 44 patients represented 0.47% of the 9,420 patients with psoriasis who were seen at our institution between 1979 and 1989. Of the 23 patients who had photosensitivity without LE, 11 were male and 12 were female; of the 21 with LE, 5 were male and 16 were female (76%).

We found 25 cases of photosensitivity among the 23 patients with psoriasis and no history of LE. The causes of photosensitivity in this group are listed in Table IV. PMLE was the most frequent cause identified, and 80% of photosensitivity cases were due to PMLE, drugs, psoralen and ultraviolet light A (PUVA), or photocontact reactions. One instance of ultraviolet light B (UVB)-induced erythema during the Goeckerman treatment was not considered true photosensitivity.

UVB alone or the Goeckerman regimen was used to treat five of the eight patients with a history of PMLE, all of whom had clearing of psoriasis. The only patient treated with PUVA who had a history of PMLE also had clearing of psoriasis. UVL was given cautiously in these patients so as to avoid phototoxic doses yet promote tanning.

Table IV. *Causes of photosensitivity among 23 patients with psoriasis and no history of lupus erythematosus*

Cause	Cases		Sex, no.	
	No.	%	Male	Female
PLME[a]	8	32	2	6
Drug RXN	5	20	3	2[b]
PUVA RXN	4	16	1[c]	3
Photocontact	3	12	3	0
Type I skin	1		1	0
Vitiligo	1		1	0
UVB[d] erythema	1		0	1
Uncharacterized	2		1	1
Total	25		12	13

[a] PMLE, polymorphous light eruption.
[b] One patient had drug reaction (RXN) and also psoralen and ultraviolet light A (PUVA) RXN.
[c] One patient had PUVA reaction (RXN) and photocontact eruption.
[d] UVB, ultraviolet light B.

Of the five cases of drug-induced photosensitivity, four were related to hydrochlorothiazide/triamterene combinations, and one was related to amiodarone. None of the five patients had been treated with PUVA near the time of their reaction (54).

Two of the four patients with episodes of PUVA reactions tolerated subsequent PUVA therapy well. The remaining two patients, one who had generalized pustular psoriasis and one who had a low titer of positive antinuclear antibody, were not re-treated with PUVA. All patients had been treated with systemically administered psoralens (50, 55, 57), and no patient with a PUVA reaction was using a photoactive medication at the time of the reaction (54). Of the 19 patients with other causes of photosensitivity, 2 had received PUVA treatment without difficulty.

Of the three cases of photocontact reactions, two were due to the use of after-shave lotion and one was due to a sunscreen. A fourth patient had a history of a PUVA reaction and a separate episode of photosensitivity presumed to be due to a deodorant soap.

One patient with uncharacterized photosensitivity had a minimal erythema dose of ultraviolet light A (UVA) of 5 J/cm^2, a minimal erythema dose of UVB of 5 mJ/cm^2, and multiple positive patch tests without obvious relevance; he tolerated cautious Goeckerman therapy up to daily UVB doses of 84 mJ/cm^2 after 36 days on one occasion, with good clearing of psoriasis. The other patient with uncharacterized photosensitivity experienced an incident of indeterminate etiology,

either UVB phototoxicity or irritation from 2% crude coal tar during natural UVL therapy at home; she also tolerated cautious Goeckerman therapy to daily UVB doses of 110 mJ/cm^2 in the hospital.

Lupus erythematosus

Of our 65 patients, 42 had psoriasis and LE; they represented 1.1% of the 3,818 patients with LE seen at our institution between 1979 and 1989. The female:male ratio was 2.2:1. The mean age at onset of LE was 39 years (range, 17 to 77) for the female patients and 47 years (range, 13 to 74) for the male patients.

The distribution of the subtypes of LE and the relationship between the onset of LE and psoriasis are shown in Table V. Patients with systemic lupus erythematosus (SLE) composed nearly half the total group. Patients in whom lupus was suspected are discussed later.

Photosensitivity was present in 21 (50%) of the 42 patients with LE. The distribution and cause of photosensitivity by subtype of LE are shown in Table VI. Of the 21 patients, 18 (72%, based on 25 cases of photosensitivity, see Table VI) had photosensitivity on the basis of underlying LE. As such, it is not surprising that photosensitivity developed with the onset of LE in 15 of these 18 patients.

Systemic lupus erythematosus

As shown in Table V, 19 patients had psoriasis and SLE, excluding those who had discoid LE (DLE) before SLE (discussed below). In the 10 patients in whom psoriasis developed first, psoriasis preceded SLE by an average of 11 years (range, 6 months to 36 years). Among the five patients with no clinical history of photosensitivity, the average interval preceding the onset of LE was 6 years, whereas among the five with photosensitivity this interval was 16 years. In the nine patients in whom SLE preceded psoriasis, the average interval was 10 years (range, 10 months to 18 years).

Table VII lists some of the characteristics of the patients with SLE (alone or preceded by DLE) who were photosensitive. Eight were photosensitive on the basis of LE.

Cutaneous manifestations were found in 14 (64%) of the 22 patients with SLE, including those with SLE subsequent to DLE, as detailed in Table VIII. Of these 22 patients, 11 were photosensitive and 11 were not. Of the 11 photosensitive patients, 9 (82%) had cutaneous manifestations. Of the 11

Table V. *Distribution of subtypes of lupus erythematosus among 42 patients with psoriasis and the chronology of onset of lupus erythematosus in relation to psoriasis*

Subtypes	Patients		Sex, no.		Chronology of onset, no. patients[a]		
	No.	%	Male	Female	Concomitant	PSOR 1st	LE 1st
SLE[b]	19	45	6	13	0	10	9
DLE[c]	7	17	2	5	1	2	4
Lupus suspect	7	17	1	6	1	6	0
Drug-induced	5	12	3	2	0	4	1
DLE/SLE[d]	3	7	1	2	1	0	2
DDLE[e]	1	2	0	1	0	1	0
Total	42	100	13 (31%)	29 (69%)	3 (7%)	23 (55%)	16 (38%)

[a] PSOR, psoriasis; LE, lupus erythematosus.
[b] SLE, systemic lupus erythematosus.
[c] DLE, discoid lupus erythematosus.
[d] DLE/SLE, DLE with subsequent SLE.
[e] DDLE, disseminated DLE.

Table VI. *Cause of photosensitivity among subgroups of 42 patients with lupus erythematosus and psoriasis*

Subtype[a]	No. of patients	No. with photosensitivity	Cause of photosentivity, no.			
			Underlying LE	PUVA RXN	Type I skin	Photo-contact
SLE	19	9[b] (47%)	6	2	3	1
DLE	7	4	4			
Lupus suspect	7	5[c]	5	1		
Drug-induced	5	0				
DLE/SE	3	2	2			
DDLE	1	1	1			
Total	42	21 (50%)	18 (72%[d])	3 (12%)	3 (12%)	1 (4%)

[a] See Table 5 for definitions of subtypes.
[b] Three patients had two different identifiable causes.
[c] One patient had psoralen and ultraviolet light A reaction (PUVA RXN) and photosensitivity on the basis of underlying lupus erythematosus.
[d] Percentages are based on 25 cases of photosensitivity.

Table VII. *Selected characteristics of patients with psoriasis, systemic lupus erythematosus, and photosensitivity*

Case, by cause of PTS[a]	Sex	Severity	Sites of PTs	Studies[b]	Associated disorders[c]	Comments[d]
SLE						
1	F	Mild	Face Arms	+ ANA, RF +RNP, UB nl ds-DNA, CH_{50}	Rheumatoid arthritis	DLE 5 yr before SLE PSOR 5 mo after PLAQ Typical cutaneous SLE
2	M	Moderate	Face Arms	+ANA, UB, ds-DNA +LAC, anti-HAV $\downarrow CH_{50}$, C_3, C_4 non-dx Bx, s/o DIF	Lupus anticoagulant syndrome	DLE 30 yr before SLE Vasculitis with digital ulcers
3	M	Mild	Face Arm Hands	+ANA, UB, ds-DNA $\downarrow CH_{50}$; nl C_3, C_4 dx Bx, dx DIF	Hypothyroidism, TTP Lichen planus-mouth, leg	PSOR within 1 yr of PLAQ Typical cutaneous SLE and livedo of legs
4	M	Mild	Face Arms Hands	+ANA nl ds-DNA, CH_{50} non-dx Bx, DIF	Avascular necrosis	Typical cutaneous SLE
5	F	Mild	Face	+ANA, ds-DNA +ss-DNA nl CH_{50}, C_3, C_4 +Renal Bx-membranoproliferative		Malar erythema and livedo of legs
6	F	Moderate	Face	+ANA, ds-DNA $\downarrow CH_{50}$, C_4; nl C_3 non-dx Bx + Renal Bx-mesangial		Guttage PSOR resembling DDLE UVB tolerated until onset of LE, then intolerant
7	F	Moderate	Face Arms	+ANA, ds-DNA, ACA nl ENA, +ss-DNA $\downarrow CH_{50}$, C_3, C_4 s/o Bx, dx DIF	Lupus anticoagulant syndrome	Typical cutaneous SLE and chronic leg ulcers
SLE and type I skin 8	F	Mild	Face Arms Legs	+ANA, ds-DNA nl CH_{50}	Pernicious anemia	Severe systemic sun rxn in Jamaica 2 yr before LE diagnosed No cutaneous LE
Type I skin and photo-contact 9	F	Moderate	Face Arms Hands Legs	+ANA, Ro, LA nl ds-DNA, CH_{50} nl porph s/o Bx, DIF UB Med+5 mJ UVA Med −1, 5 J +Photopatch to bergamot	Inflammatory bowel disease	UVB aggravated PSOR and LE after onset LE PLAQ aggravated PSOR Procainamide ×3 yr, until 2 yr before onset LE Typical cutaneous SLE
Type I skin and PUVA RXN 10	F	Mild	Face Arms	+ANA nl CH_{50} ± ds-DNA UVA Med no RXN non-dx DIF		PUVA RXN once Tolerated cautious Goeckerman and PUVA otherwise NO cutaneous LE
PUVA RXN 11	F	Moderate	Face Chest	+ANA, ds-DNA $\downarrow CH_{50}$, C4 dx, Bx, DIF +anti-HAV	Carpal tunnel syndome	PUVA aggravated SLE Typical cutaneous SLE

[a] PTS, photosensitivity; SLE, systemic lupus erythematosus; PUVA RXN, psoralen and ultravoilet light A reaction.
[b] ANA, antinuclear antibody; RF, rheumatoid factor; RNP, ribonucleoprotein; UB, unidentified band, ds-DNA, double-stranded DNA; CH_{50}, total hemolytic complement; LAC, lupus anticoagulant, anti-HAV, antibody to hepatitis A virus; C_3, third component of complement; C_4, fourth component of complement; non-dx, nondiagnostic; Bx, biopsy; s/o, suggestive of; DIF, direct immonofluorescent biopsy; dx, diagnostic; ss-DNA, single-stranded DNA; ACA, anticardiolipin antibody; ENA, extractable nuclear antigens; Ro, Ro/SSA antibody; La, La/SSB antibody, porph, porphyrin; UVB Med, ultraviolet light B, minimal erythema dose; UVA Med, ultraviolet light A, minimal erythma dose; +, positive or elevated; nl, negative or normal; −, low; ±, borderline.
[c] TTP, thrombotic thrombocytopenic purpura.
[d] DLE, discoid lupus erythematosus; PSOR, psoriasis; PLAQ, Plaquenil (hydroxycloroquine sulfate); DDLE, disseminated DLE.

Table VIII. *Distribution of cutaneous involvement in 22 psoriatic patients with SLE, including those with SLE subsequent to DLE*[a]

Site	Patients ($n=22$)		Photosensitive patients ($n=11$)		Nonphotosensitive patients ($n=11$)	
	No.	%	No.	%	No.	%
Scalp	2	9	1	9	1	9
Head/neck	6	27	4	36	2	18
Malar	6	27	4	36	2	18
Oral	2	9	1	9	1	9
Extremity	6	27	4	36	2	18
Trunk	8	36	5	45	3	27
Dorsal hand/forearm	3	14	2	18	1	9
Alopecia	5	23	3	27	2	18
Other	4	18	3[b]	27	1[c]	9
None	8	36	2	18	6	55

[a] SLE, systemic lupus erythematosus; DLE, discoid lupus erythematosus.
[b] Two patients with livedo of legs, one with vasculitic digital infarct.
[c] Perineal erosions.

patients without photosensitivity, only 5 (45%) had cutaneous manifestations.

In general, the cutaneous manifestations were typical of SLE in these 14 patients and remained distinct from lesions of psoriasis. However, three patients (21%) had some clinically atypical or overlap morphology of lupoid and psoriasiform lesions. One patient (case 6 in Table VII) had generalized guttate psoriasiform lesions that resembled disseminated DLE; a previous skin biopsy was diagnostic of psoriasis and a recent biopsy of a suspected lupoid lesion was nondiagnostic. In a second patient with noncutaneous SLE, a recurrent atypical erythematosquamous scalp plaque developed 8 years after the onset of SLE; this provided a biopsy diagnostic of psoriasis. Subsequently, typical plaques of psoriasis developed elsewhere. In the third patient, a psoriasiform eruption developed; biopsy was not performed, but the eruption responded to topically applied steroids and topically applied tar preparations. Four years later the patient was noted to have an "erythematous, scaling, nonatrophic dermatitis" over the trunk and malar areas, "seborrheic versus lupoid." A psoriasiform plaque was noted over the left elbow, in addition to gluteal cleft erythema and scaling. Two lesional biopsy specimens from the cheek and chest were consistent with seborrheic dermatitis, but a direct immunofluorescent biopsy specimen from the chest lesion was diagnostic of LE.

Fourteen lesional biopsy specimens were obtained from 10 of the 14 patients with SLE who had cutaneous disease. Routine histology was diagnostic in only three patients (21%) and suggestive in three (21%). The remaining 8 biopsy specimens were nondiagnostic. Immunofluorescence testing of 23 biopsy specimens from the 14 patients was diagnostic or suggestive of LE in 10 (43%) of the specimens. Interestingly, 7 (58%) of the 12 biopsy specimens from the photosensitive group were diagnostic or suggestive of LE, whereas only 3 (27%) of the 11 specimens from the nonphotosensitive group were diagnostic of LE.

There were no significant differences in systemic involvement by SLE between the photosensitive patients and the nonphotosensitive patients (Table IX).

Laboratory evaluation of the patients with SLE revealed positive antinuclear antibody tests in 100%. One or more

Table IX. *Systemic involvement in 22 patients with systemic lupus erythematosus and psoriasis*

System affected	Photosensitive patients ($n=11$)		Non-photo sensitive patients ($n=11$)	
	No.	%	No.	%
LE-associated joint	7	64	6	55
Hematologic	7	64	9	82
Renal	4	36	5	45
Neurologic	2	18	2	18
Serositis	4	36	5	45
Raynaud's	4	36	2	18
Other	4[a]	36	3[b]	27

[a] One each with lupus anticoagulant syndrome, pulmonary fibrosis, vasculitic peripheral neuropathy, and deep venous thrombosis with thrombophlebitis.
[b] One each with subcutaneous and pleural calcification, interstitial fibrosis; central retinal vein obstruction; pneumonitis, restrictive lung disease, and myelitis with cord infarct.

extractable nuclear antigens (Ro, La, ribonucleoprotein, Sm, or "unidentified bands" [bands reported before our laboratory began reporting Ro or La]) were positive in four of the six photosensitive patients tested but in only one of the seven nonphotosensitive patients. The level of antibodies to double-stranded DNA was elevated in 7 (64%) of the 11 photosensitive patients and in only 1 (9%) of the 11 nonphotosensitive patients (3 of the 11 patients in this group had borderline values). No significant differences were noted in level of complement, sedimentation rate, rheumatoid factor, immunoglobulins, or positive results of rapid plasma reagin tests.

Discoid lupus erythematosus

Eleven of our patients had DLE (seven had localized disease, one had disseminated disease, and three had DLE before SLE). The mean duration of disease among these patients was

19 years. Seven of these patients were photosensitive, all on the basis of underlying LE. The distribution of lesions was as follows: scalp, six; head and neck (excluding scalp), five; extremities, four; malar, three; trunk, three; oral, one. As indicated in Table V, discoid lesions preceded psoriasis in six patients; the average interval was 15 years (range, 7 to 24 years) (interval was uncertain in one patient). The SLE that subsequently developed in two of these patients preceded psoriasis by 1 and 14 years. Psoriasis preceded DLE in three patients; the average interval was 23 years (range, 4 to 46 years). In two patients, DLE and psoriasis developed concomitantly.

Four of the 11 patients with DLE had some atypical presentation. In one patient with a single right thigh plaque, routine histology was diagnostic of psoriasis, and direct immunofluorescent biopsy was diagnostic of LE; typical scalp psoriasis developed 14 years later. One patient had a 10-year history of an "atypical, seasonal, PMLE-like, photokoebnerized eruption" on the face and scalp; routine and direct immunofluorescence biopsy results were diagnostic of LE. Biopsy-proven localized and then generalized pustular psoriasis developed 15 years after DLE was diagnosed.

In another patient, who had a 46-year history of severe psoriasis, an atypical blistering eruption of the scalp, forehead, and oral mucosa developed, and scaling erythema and erosions were present on the abdomen. Routine testing of exposed, lesional biopsy specimens was nondiagnostic; however, direct immunofluorescence testing of unexposed, lesional skin was suggestive of LE, with granular C3 and fibrinogen at the basement membrane zone, linear IgG at sweat gland basement membrane zone, and cytoids staining with IgM.

In the fourth patient, who had a 20-year history of psoriasis, psoriasiform photo-accentuated plaques developed on the scalp, extremities, and trunk during Goeckerman therapy for psoriasis. Routine histology was diagnostic of LE, but direct immunofluorescence was negative. She was found to have positive antinuclear and Ro antibodies. She had no evidence of SLE and was originally considered as having disseminated DLE, but the findings better fit the category of subacute cutaneous LE.

Routine histology of nine specimens from six patients with DLE was diagnostic in six and suggestive in one. Direct immunofluorescence of nine specimens from seven patients was diagnostic of LE in five (lesional) and suggestive in one (lesional). Two of the three negative specimens were from uninvolved skin.

In the eight patients with DLE without subsequent SLE, positive antinuclear antibodies were found in seven. Of three patients tested, one each had positive antibodies to Ro, ribonucleoprotein, and an unidentified band. The erythrocyte sedimentation rate was elevated in four of the eight patients, and there were no significant abnormalities of complement, double-stranded DNA, rapid plasma reagin, rheumatoid factor, or serum protein electrophoresis.

Suspected lupus

Seven patients initially did not meet the American Rheumatism Association criteria for a diagnosis of LE but had certain features suggesting this diagnosis. Pertinent aspects of these cases are listed in Table X. Five of these seven patients had photodistributed psoriasis or an abnormal response to UVL. The two remaining patients had no photosensitivity and no atypical cutaneous lesions.

One patient (case 1) had a 2-year history of biopsy-proven psoriasis and presented with plaques over the extensor arms; the central part of the face was spared and nonphotodistributed areas were involved (Fig. 1 and 2). Routine histology from a plaque on the upper aspect of the mid-back (Fig. 3) was interpreted as a "psoriasiform dermatitis," and direct immunofluorescence of the same plaque was diagnostic of LE. Routine and Hep-2 antinuclear antibodies were then found to be positive in low titer, and anti-Ro antibody was present. The patient had been previously intolerant of methotrexate and wanted to become pregnant. The minimal erythema dose of UVB was 13 mJ/cm^2. She was treated in a hospital setting with the Goeckerman regimen. Suberythermal daily doses of UVB were given cautiously and were increased to maximal doses of 11 mJ/cm^2 after 22 treatments during one hospitalization and to 12 mJ/cm^2 after 18 treatments during a second hospitalization 2 years later. Her psoriasis remitted in each instance without adverse effects.

Another patient (case 2) presented with a 10-year history of scalp psoriasis and atypical malar, arm, hand, and chest lesions before development of an erythrodermic flare of psoriasis. Routine histology from the right dorsal aspect of the hand (Fig. 4) was diagnostic of psoriasis, and direct immunofluorescence of the same site was diagnostic of LE. Evaluation then revealed positive antinuclear antibodies and antiribonucleoprotein antibodies. Intolerant of dapsone, this patient was treated with topically applied steroids and tar preparations; improvement was modest. No further follow-up data were available after her initial treatment.

The third patient (case 3) had a 17-year history of psoriasis that was previously responsive to UVB and a modified PUVA regimen before development of intolerance to UVL. Atypical papulosquamous lesions on the dorsal aspect of the hands were suggestive of LE in 1979 (Fig. 5). Erythroderma developed with UVL and tar treatments at her home institution. Skin biopsy specimens from the thigh and left third finger (Fig. 6) were diagnostic of psoriasis, and direct immunofluorescence testing of involved skin of the calf and left third finger was suggestive of LE, as was testing of uninvolved skin of the left hip. The patient had positive antinuclear antibody and anti-Ro antibodies with an associated C2 deficiency. Her psoriasis improved with methotrexate, but its use was then discontinued because of leukopenia. Her condition was uncontrolled with hydroxychloroquine, and she was maintained on dapsone and had flaring only with coincident infections.

In case 4, the patient had a 13-year history of psoriasis that was previously responsive to UVB and PUVA. He repeatedly denied any history or symptoms of photosensitivity, and he had no symptoms or signs of LE. He was admitted for the Goeckerman treatment and responded well, although he had persistent prominent erythema within and limited to resolving psoriatic plaques. Antinuclear antibody was positive, as were anti-Ro and La antibodies and an unidentified band. His treatment regimen was completed with excellent results and no complications; phototoxicity was avoided by giving daily suberythemal amounts of UVB.

Case 5 has been described previously (25). The patient had a 15-year history of psoriasis that was unchanged by UVB therapy in the past. She presented with atypical photodistributed plaques and an initial diagnosis of disseminated DLE

Table X. *Chracteristics of seven patients with psoriasis in the whom lupus was suspected*

Case[a]	Sex	Features of lupus erythematosus	Studies[b]	Associated disorders	Comments[c]
			Photosensitive		
1 Photo-dist PSOR Seasonal Erythema	F	Photosensitive synovitis	BX, psor derm DIF, LE +ANA, 1:20, Hep-2 +Ro nl CH_{50}, ds-DNA UVB Med, 13 mJ		Tolerated cautious suberythemal Goeckerman
2 Erythrodermic flare of PSOR	F	Atypical malar, arm, hand, chest lesions Atypical arthritis, ?rheumatoid arthritis Raynaud's	Bx, PSOR DIF, LE +ANA, Hep-2 ±ds−DNA nl, CH_{50}, porph +RNP UVB Med, 7 mJ UVA Med, no RXN +RF, low titer	? rheumatoid arthritis	'Tanning booth' had improved PSOR in past
3 Recent intolerance to sun	F	Photosensitive, atypical hand lesions	Bx, PSOR DIF, s/o LE +ANA, Ro ±ds-DNA ↓CH_{50}, C_2; nl $C_{3,4,5}$ Leukopenia Occ proteinuria	C2 deficiency	Tolerated cabinet ultraviolet light to 7½ min. before LE onset
4 Erythema in resolving PSOR plaques during Goeckerman	M	None	+ANA, Hep-2 nl ds-DNA, CH_{50} +Ro, LA, UB Lymphopenia		Tolerated ultraviolet light B, PUVA, Goeckerman
5 Photo-dist PSOR PUVA-induced erythroderma	F	Atypical photo-dist plaques	Bx, PSOR DIF, LE −ANA, Hep-2 ±ds-DNA ↓CH_{50}, $C_{2,3,4}$ nl C_1Ei Lymphopenia	C2 deficiency with urticaria/angioedema in mother	Erythrodermic after 10 PUVA treatments
			Nonphotosensitive		
6 None	F	Atypical arthritis	−DIF +ANA, high titer −La, RNP, Sm nl CH_{50} −HLA−B27		Toleratred ultraviolet light B
7 None	F	Synovitis Atypical arthralgias	+ANA −La, RNP, Sm nl ds-DNA, CH_{50}	Nontropical sprue	Tolerated ultraviolet light B

[a] Photo-dist, photo-distributed; PSOR, psoriasis; PUVA, psoralen and ultraviolet light A.

[b] Bx, biopsy, psor derm, psoriasiform dermatitis; DIF, direct immunofluorescent biopsy; LE, lupus erythematosus; ANA, antinuclear antibody; Ro, Ro/SSA antibody; CH_{50}, total hemolytic complement; ds-DNA, double-stranded DNA; UVB Med, ultraviolet light B, minimal erythema dose; porph, porphyrin; RNP, ribonucleoprotein; UVA Med, ultraviolet light A, minimal erythema dose; RXN, reaction; RF, rheumatoid factor; s/o, suggestive of; C_2, second component of complement; C_3, third component of complement; C_4, fourth component of complement; C_5, fifth component of complement; Occ, occasional; La, La/SSB antibody; UB, unidentified band; C_1Ei,C_1 esterase inhibitor; Sm, Smith antigen; +, positive or elevated; nl, negative or normal; −, low; ±, borderline.

[c] PUVA, psoralen and ultraviolet light A.

in 1976; routine histology was diagnostic of psoriasis, but direct immunofluorescence was diagnostic of LE (exposed, lesional skin). Evaluation revealed an associated C2 deficiency with negative antinuclear antibody and Hep-2 antinuclear antibody and borderline antibodies to double-stranded DNA. Another evaluation for PUVA-induced erythroderma in 1985 provided another routine biopsy diagnostic of psoriasis and direct immunofluorescence diagnostic of LE (exposed, lesional). She has been maintained with topically applied steroids and topically applied tar preparations.

In the two remaining patients (cases 6 and 7), lupus was suspected on the basis of persistent atypical arthralgias with positive antinuclear antibodies. In both cases, careful UVB treatments had been used in addition to other regimens; there was good clearing of psoriasis, and there were no adverse effects.

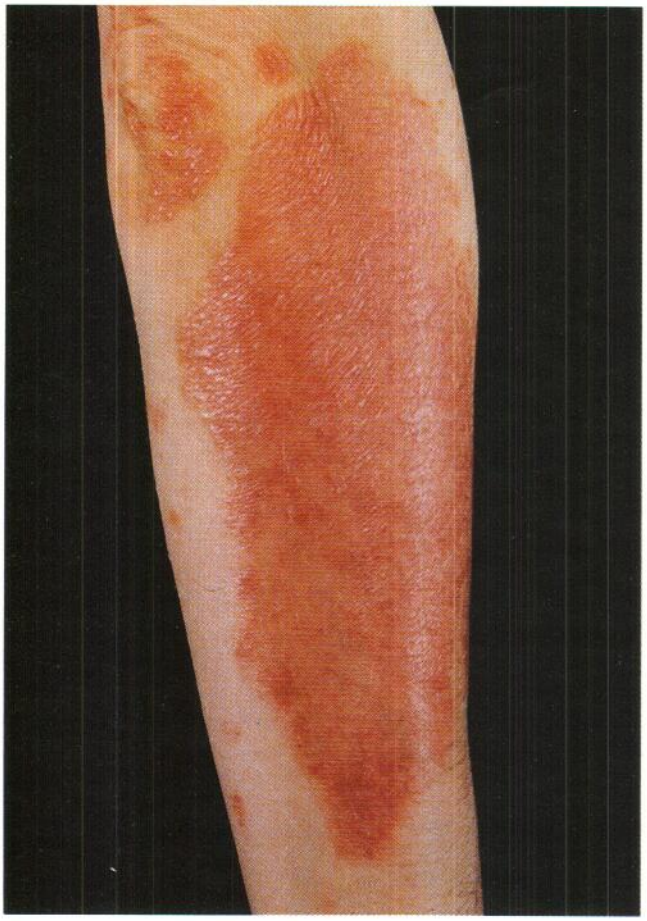

Fig. 1. (case 1, Table X). Thin plaques of psoriasis involving extensor forearm.

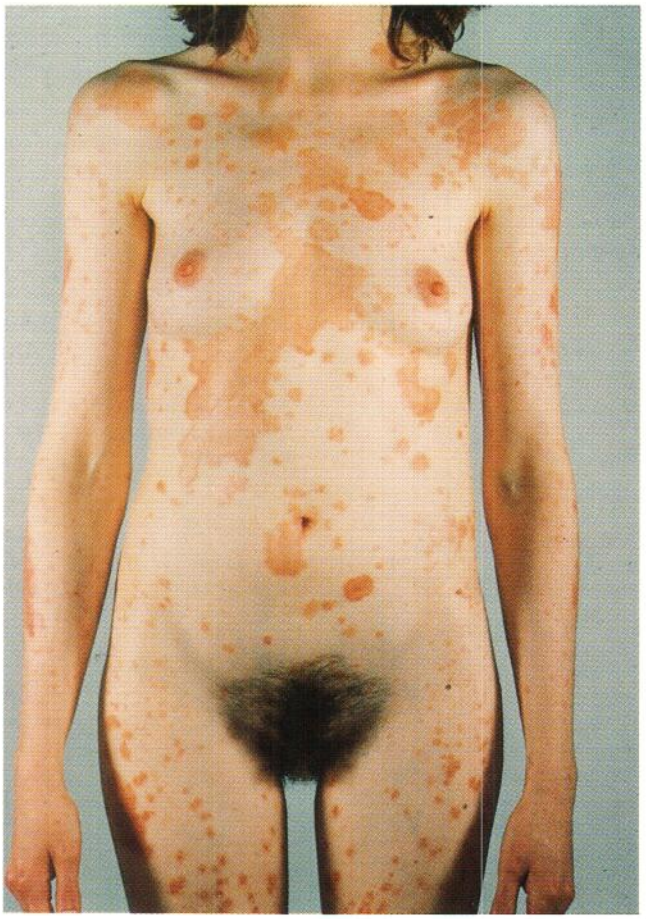

Fig. 2. (case 1, Table X). Generalized thin, large, guttate plaques of psoriasis involving exposed and unexposed portions of trunk and extremities.

Drug-induced lupus erythematosus

The five patients with drug-induced LE and psoriasis had no cutaneous lesions of LE and no history of photosensitivity. Four cases were due to procainamide and one to phenytoin. Antinuclear antibodies were positive in four.

Treatment

Controlled trials were not performed in these patients, but the following is a summary of the treatments used and the results.

Topically applied steroids were used in 63 of the 65 patients; they were well tolerated but were of limited usefulness.

Systemically administered steroids were used in 35 patients, 29 of whom had LE. Psoriasis was unaffected in 18 patients, improved in 16, and initially developed after taper of the prednisone dose in 1.

Topically applied tar preparations were used in 38 patients: psoriasis improved in 32, did not change in 2, and was aggravated in 4 (all with LE). Lesions of LE were aggravated in three patients.

UVB alone was used in 22 patients, and psoriasis improved in 16. This treatment improved psoriasis but aggravated LE in one patient and aggravated both psoriasis and LE in three patients (two of whom had initially improved), one each with SLE, disseminated DLE, and suspected lupus. In no case was the suspected aggravation of LE a serious problem, and its management was straightforward.

The Goeckerman regimen was used in 19 patients. Sixteen patients responded well, five with "cautious" regimens (one with SLE, one Ro-positive results and suspected lupus, two with type I skin, and one with a minimal erythema dose of 5 J/cm^2 for UVA and of 5 mJ/cm^2 UVB). Psoriasis was aggravated in one patient while receiving trimethoprim/sulfamethoxazole, LE was aggravated in one, and psoriasis and LE were both aggravated in one patient after initial tolerance.

PUVA was used in 11 patients, 8 of whom responded well. The treatments were discontinued in one patient because of increased liver enzyme values. The psoriasis was aggravated by PUVA in one patient, LE was aggravated in one patient, and both conditions were aggravated in one patient. Of four patients without LE who had reactions to PUVA, two tolerated subsequent PUVA, and two were not rechallenged. Of two patients with SLE and PUVA reactions, one (case 10, Table VII) had no cutaneous LE and tolerated subsequent PUVA, and one (case 11) remained intolerant to PUVA on rechallenge by her local doctor. One patient (case 5, Table X) in whom lupus was suspected was not rechallenged after the development of PUVA-induced erythroderma during treatment by her local doctor.

Antimalarials were used in 20 patients: 17 had improvement

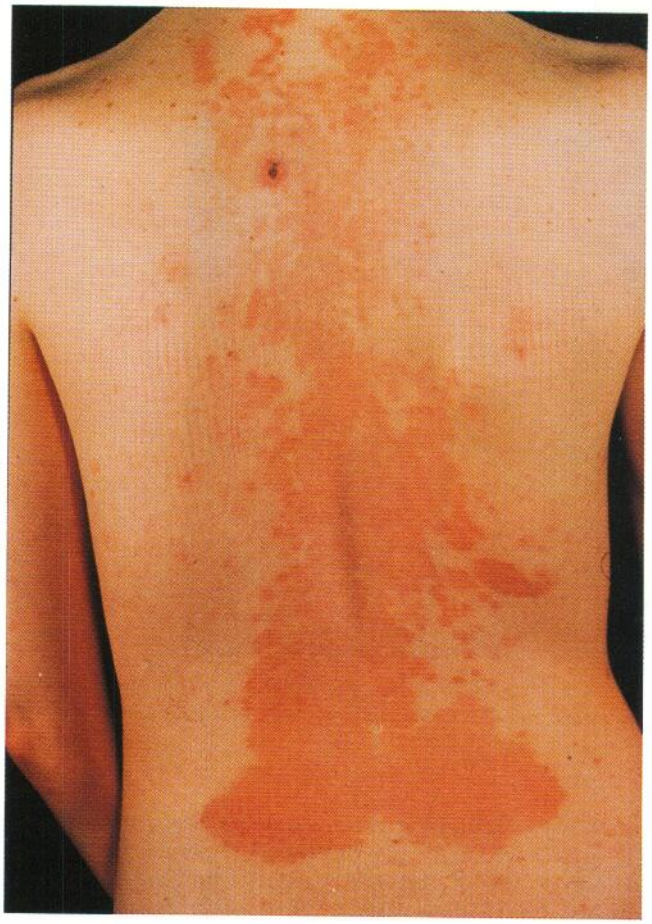

Fig. 3. (case 1, Table X). Large, psoriasiform plaque of back. Routine histology was interpreted as "psoriasiform dermatisis," and direct immunofluorescence from same biopsy site was diagnostic of lupus erythematosus.

of LE and 3 had no effect. Psoriasis was aggravated in five patients: in two psoriasis developed within 1 year of beginning the use of hydroxychloroquine, and three experienced an aggravation of preexistent psoriasis. Cytotoxic medications, primarily methotrexate, were used in 17 patients: 13 experienced improvement of psoriasis and 4 noted no effect. Of eight patients with lupus, one experienced improvement in arthritis and seven were unaffected. Side effects necessitating discontinuation of treatment included leukopenia or thrombocytopenia (two patients), elevated liver enzyme values (one), oral ulcers (two), and nausea (two).

Nonsteroidal anti-inflammatory medications were prominently used, usually for arthralgias or arthritis, in 17 patients: 11 had improvement in LE-related symptoms, 5 had improvement in psoriasis-related symptoms, and 1 had no effect. No adverse effects were noted.

Retinoids were used in seven patients: five had improvement in psoriasis, one had no effect, and one patient sensitive to UVA had aggravation of psoriasis. No aggravation of LE was noted.

Gold was used in six patients: three had improvement in psoriasis, and three had no change. Glomerulonephritis developed in one patient. Dapsone helped psoriasis and LE in one of two patients. Azathioprine benefited LE in two patients, and there was no change in psoriasis. Cyclosporine, vitamin D_3, and chlorambucil improved the psoriasis in one patient each without affecting the LE.

DISCUSSION

Estimates of the prevalence of photosensitivity among patients with psoriasis vary from 5.5% to 24% (59–63, 65, 67, 68). The estimate of 5.5% by Ros et al. (66–69) may be the most accurate to date; it was derived from 1,760 respondents to a questionnaire and interview after exclusion of patients with photoirritable psoriasis and concomitant dermatoses (67).

The present study is an attempt to evaluate patients who have psoriasis and photosensitivity of any cause, including those with coexistent LE. Photosensitivity was defined as any adverse cutaneous eruption due to exposure to UVL. Of 9,420 patients with psoriasis, only 65 (0.69%) had coexistent photosensitivity or LE, including 21 with LE but no photosensitivity. The 44 patients with some history or evidence of photosensitivity represented less than 0.5% of patients with psoriasis who were seen during the 10-year study period; this estimate is significantly lower than those reported previously. This difference may be due in part to the retrospective nature of the review because charts were initially screened on the basis of dismissal diagnoses. Screening would not have identified patients who may have had evidence of photosensitivity but in whom it was not prominent enough to warrant a specific dismissal diagnosis. However, with an average follow-up period of 16 years, significant photosensitivity should have been noted at some point. Therefore, although our estimate may be artificially low, it is doubtful that a large number of patients with significant photosensitivity were left unidentified.

Psoriasis

The characteristics of psoriasis in our patients do not substantially differ from those of patients with psoriasis who do not have photosensitivity or LE, except for an increased frequency of erythroderma. At least one episode of subsequent erythroderma was found in 7 (13%) of the 56 patients with plaque-type disease, in 3 of whom lupus was suspected (1 was Ro-positive, 1 was ribonucleoprotein-positive, and 1 had a C2 deficiency with PUVA-induced erythroderma). This frequency is comparable to instances of erythroderma in 1 of the 11 patients with psoriasis and LE reported by Lynch & Roenigk (23) and in 4 of the 27 described by Millns & Muller (26).

Of our patients treated with antimalarials for LE, 25% had new-onset psoriasis or aggravation of preexisting psoriasis. Estimates of exacerbation of psoriasis by antimalarials vary. Cornbleet (70) noted exacerbation in six of six patients treated with antimalarials. Bielicky (71) found that 8 of 10 patients who had psoriasis without photosensitivity had an exacerbation with chloroquine treatment, whereas none of the 14 "light-sensitive" patients experienced worsening; this finding raises the question of whether these latter patients may have had LE. In another study (72), Bielicky et al. found that 4 (22%) of 18 patients deteriorated with chloroquine treatment, a finding similar to ours. O'Quinn et al. (73) noted no exacerbation in nine patients with psoriasis who were treated with chloroquine, but they found increased light sensitivity and decreased minimal erythema dose values for UVB in six of the nine patients. Baker (74) had to discontinue antimalarial

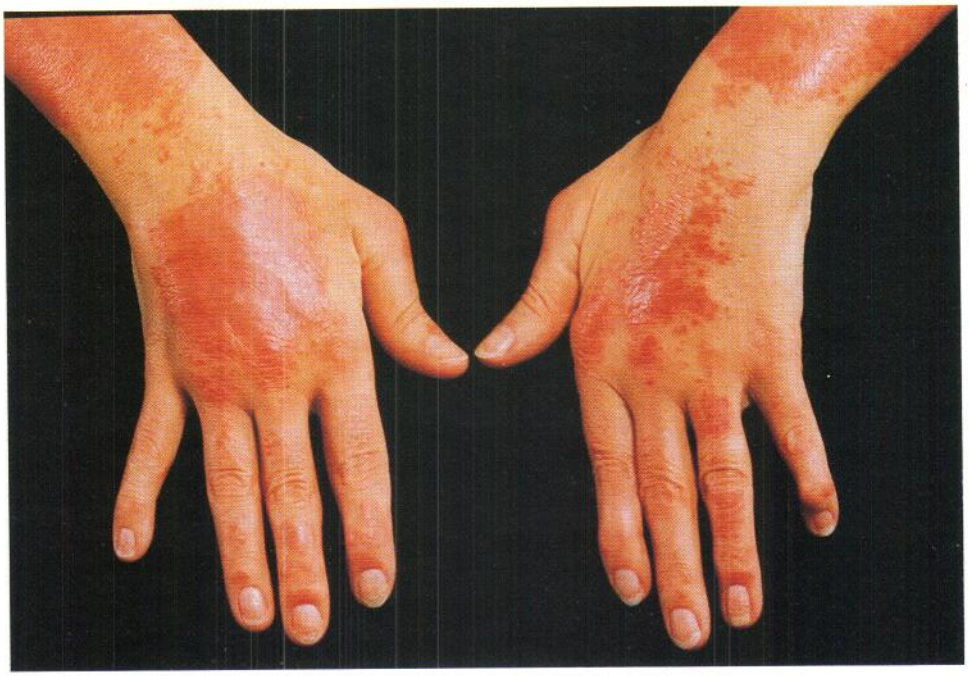

Fig. 4. (case 2, Table X). Psoriasiform plaques involving extensor forearm, dorsal hand, and dorsal phalangeal skin. Note onycholysis and pitting of nails. Routine histology of right dorsal hand was diagnostic of psoriasis, and direct immunofluorescence of same site was diagnostic of lupus erythematosus.

treatments in one of six patients because of aggravated psoriasis. One of 11 cases reported by Lynch & Roenigk (23) may have been precipitated by hydroxychloroquine. Fifty patients treated by Kammer et al. (75) tolerated hydroxychloroquine for psoriatic arthritis without a flair of cutaneous disease. However, 20 (42%) of the 48 patients described by Kuflik (76) noted exacerbation of psoriasis during treatment with various regimens of antimalarials for prophylaxis while in Vietnam, and treatment resistance developed in 3. Millns & Muller (26) noted aggravation in one of four patients with antimalarial treatment. Thus, there may be a significant risk of aggravating psoriasis with antimalarial therapy in photosensitive patients.

Photosensitivity

The 23 patients with psoriasis and some history of photosensitivity but no history of LE represented only 0.24% of the patients with psoriasis who were seen during the decade-long study. The mean age of this group was 63 years. Of the 23 patients, 6 (26%) had type I skin. The latter findings are similar to those of Ros & Eklund (67). Fewer of our patients (23%) had psoriasis involving the hands.

PMLE was the cause of photosensitivity in 32% of these 23 patients, and in the study by Ros & Eklund (67) it was the cause in 43% of photosensitive patients with psoriasis and 35% of the nonphotosensitive patients with psoriasis who reported a history of photosensitivity (that is, those in whom psoriasis did not worsen despite photosensitivity). This frequency is somewhat higher than the 10% to 20% estimate of PMLE in the general population (66, 77).

Ros & Wennersten (68) found that PMLE was most easily produced with high-dose UVA in photosensitive patients with psoriasis. However, PUVA was efficacious in prophylaxis of PMLE in this group and in the treatment of existing psoriasis (69). Only one of eight patients was specifically treated at our institution for PMLE, and this patient responded well to UVB. However, five of the eight patients who had PMLE were treated for psoriasis with UVB alone or the Goeckerman regimen, and one patient was treated with PUVA. All tolerated the treatments well and had clearing of the psoriasis.

An accurate diagnosis of PMLE need not exclude treatment with UVB or PUVA, but special effort should be made to exclude LE as an associated diagnosis.

Lupus erythematosus

This report represents the largest series of patients to date with coexistent psoriasis and LE. The 42 patients in this group represent 1.1% of the patients with LE seen at the Mayo Clinic during the study period, and our findings agree with the observation by DuBois (78) that psoriasis occurred in 0.6% of 520 patients with LE. This coexistence probably represents a chance association, but it creates significant therapeutic implications.

In the largest studies to date, Tumarkin et al. (29) described four men and two women each with lesions of DLE preceding and remaining distinct from psoriasis. Lynch & Roenigk (23) described eight patients with SLE (six female, two male) and three with DLE (all male) with coexistent psoriasis. In eight of their patients, psoriasis developed before LE, and two patients died as a result of SLE. Apparently none of the patients had been treated with UVL before the development of LE. Millns & Muller (26) described 27 patients from our institution who had coexistent psoriasis and LE: 10 had SLE (9 female, 1 male), 13 had DLE (6 female, 7 male), and 4 had drug-induced LE or lupus-like syndromes (2 female, 2 male). Of those patients, psoriasis preceded LE in 10 patients and was concomitant with it in 6. A morphologic overlap and a

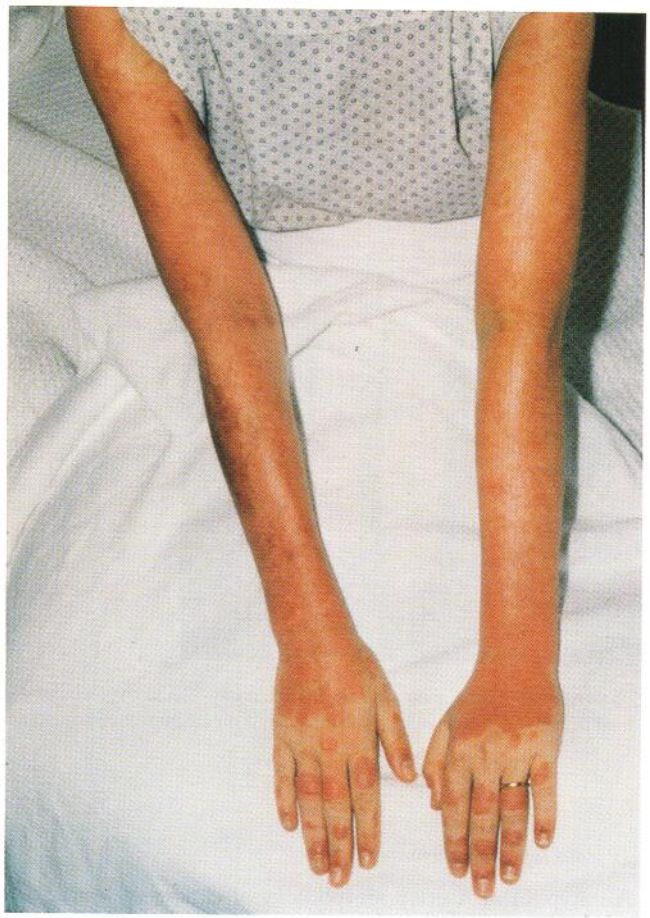

Fig. 5. (case 3 in 1979, Table X). Confluent, thin, psoriasiform plaques involving entire extensor arms, with prominent involvement of skin over phalanges. Note periungual involvement and onycholysis.

clinical interaction occurred in 7 of their patients "in whom the disorders were severe and complicated by a third disease." Eight-five percent of Millns & Muller's patients had some history of photosensitivity, which developed with LE in approximately 50%. No information was available regarding skin type or minimal erythema dose for these patients.

The current study found a predominance of patients with SLE (45%) and a predominance of females (2.2:1) in all subtypes of LE except drug-induced LE. These results are consistent with those of other studies of LE without accompanying psoriasis (79–81).

The mean age at onset of LE in our study was somewhat later than that reported in most studies of LE (79–81): 37 years for female patients with SLE, 51 years for male patients with SLE, 46 years for female patients with DLE, and 32 years for female patients in whom lupus was suspected. This difference may be due in part to small sample size. However, in several patients with long-standing nonspecific complaints (such as arthralgias), diagnosis was delayed until specific symptoms, signs, or laboratory findings developed, and thus the "age at onset" was artificially delayed.

Although estimates vary, our findings of photosensitivity in 57% of patients with DLE and 50% of patients with SLE are similar to those reported for DLE (80) and are slightly higher than those reported for SLE (82, 83).

Multiple reports have described the onset of LE or LE-like syndromes after UVL therapy for psoriasis, although causal relationships have not been clearly proved (9, 11, 18, 25). Photodistributed psoriasis has also been reported without evidence of LE or other photodermatoses in a patient with psoriasis who was sunburned but did not show other evidence of nonspecific koebnerization (84). Similar exposure to intense UVL was implicated in 26% of the photosensitive patients with psoriasis but not LE who were described by Ros & Eklund (67).

Among our patients in whom LE developed after the onset of psoriasis (excluding drug-induced LE), 7 of the 11 photosensitive patients had received prior UVL therapy, and 1 of the 8 nonphotosensitive patients had received prior UVL therapy. LE is considered a syndrome of multifactorial cause (85), and DLE and SLE represent part of a spectrum (79, 80). It is well known that UVL may induce or exacerbate cutaneous or systemic symptoms or signs of SLE (82). Whether it alone is sufficient to cause LE is unknown; however, mechanisms exist whereby UVL may be one causative factor. It causes the formation of thymine dimers and antigenic changes in DNA of skin (86, 87), and it can induce formation of antinuclear antibodies that may cross-react with native nonirradiated DNA (88). Natali & Tan (89) showed that mice sensitized to UVL-irradiated DNA and subsequently exposed to UVL developed skin lesions histologically and immunopathologically (lupus band) similar to those of SLE. UVL-denatured DNA in the nuclei of these mouse keratinocytes was extruded from the cells, diffused back across the dermoepidermal junction, and remained there or entered the circulation (89, 90). In this way, epidermal DNA antigens could gain access to the circulation. Additionally, mice previously sensitized to UVL-irradiated DNA develop glomerular immune complexes if given irradiated DNA; this result leads to the possibility of immune complex-mediated LE nephritis (91).

There is also a relationship between photosensitivity and anti-Ro (SSA) antibodies. Several studies have noted a high incidence of photosensitivity among subsets of patients positive for this antibody, including those with subacute cutaneous LE (92) and antinuclear antibody-negative LE (93). In some studies (94–96), UVL irradiation caused cultured keratinocytes to express Ro (SSA), ribonucleoprotein, and Sm antigens on their cell surfaces, whereas no cell surface expression of double- or single-stranded DNA or histone antigens was found. Deposition of anti-SSA IgG but not double-stranded DNA or Sm antibodies was seen on basal keratinocytes of human skin grafted to nude mice after intravenous infusion of antibodies. This deposition was augmented by UVB irradiation of the grafted sites (94–96).

These studies at least partially implicate UVL in the pathogenesis or expression of cutaneous LE and may explain the onset of expression of cutaneous LE in patients with psoriasis after therapy with UVL. Although our groups are small, the longer duration of psoriasis before the development of SLE in photosensitive patients (16 years, and more of these had been treated with UVL) compared with the duration in nonphotosensitive patients (only 6 years) may suggest a role for UVL and the photosensitivity related to SLE. However, this possibility does not help explain the coexistence of LE in patients with psoriasis who have not had significant exposure to UVL or in those in whom LE develops before the onset of psoriasis. This explanation likely resides in the multifactorial

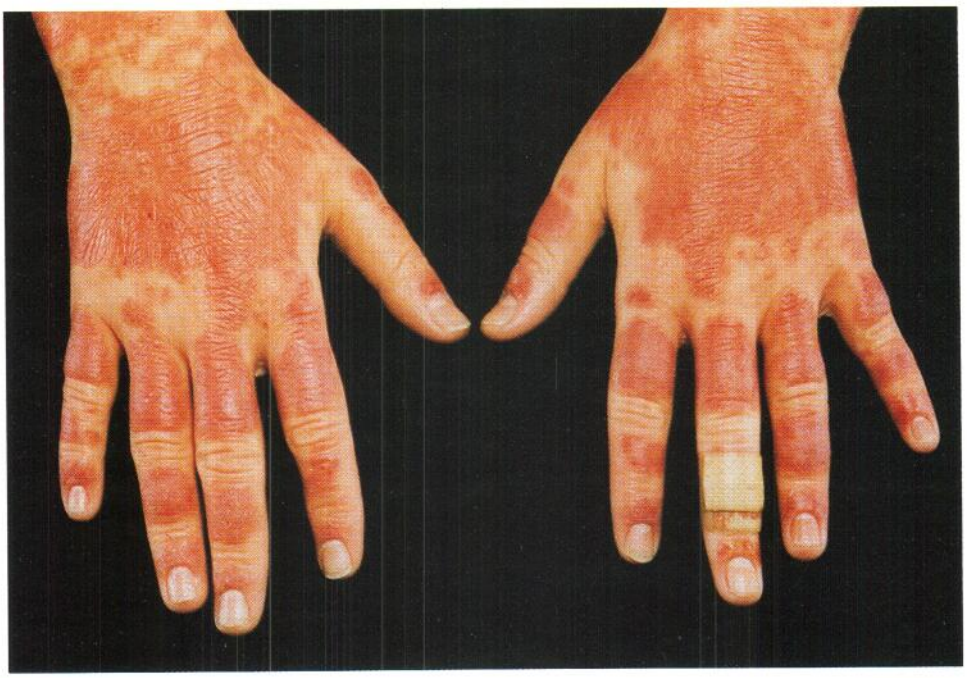

Fig. 6. (case 3 in 1985, Table X). Erythematous plaques over dorsal aspect of wrists and hands, with striking involvement of skin over phalanges. Note periungual involvement and nail pitting. Routine histology from left third finger was diagnostic of psoriasis, and direct immunofluorescence from same site was suggestive of lupus erythematosus.

nature of both processes. HLA studies in these patients may be helpful.

Systemic lupus erythematosus

The presence of cutaneous manifestations in 64% of our patients with SLE is similar to the estimates of 67% to 85% in some studies of SLE alone (82). Although lesions of LE generally remained distinct from those of psoriasis, 3 (21%) of 14 patients with cutaneous SLE had clinically atypical or overlap morphology, 2 of whom had psoriasiform eruptions and could be considered clinically to have subacute cutaneous LE, although all 3 met the criteria for the diagnosis of SLE. Only one of these patients was tested for the presence of extractable nuclear antigens, and none were found.

Biopsy specimens for direct immunofluorescence were less commonly diagnostic of either SLE or DLE than has been reported in patients who have LE without psoriasis (80). The presence of 1) extractable nuclear antigens, including Ro, in 4 of the 6 photosensitive patients with SLE but in only 1 of the 7 nonphotosensitive patients and 2) elevated levels of antibody to double-stranded DNA in 7 of the 11 photosensitive patients but in only 1 of the 11 nonphotosensitive patients with SLE suggests that, although the numbers are small, antibodies to Ro/SSA and double-stranded DNA may be associated with photosensitivity in patients with SLE and psoriasis (92, 93, 97).

It must be emphasized that laboratory test results may vary widely during the course of disease in patients with LE, and subsequent testing over time may be required in patients with psoriasis and photosensitivity.

Discoid lupus erythematosus

An atypical or overlap morphology of cutaneous lesions was found in 4 of the 11 patients with DLE. In two of these patients, direct immunofluorescence was required to confirm the diagnosis of LE because routine histology was not diagnostic or was consistent with psoriasis. This observation highlights the need for both routine and direct immunofluorescence biopsy in patients with psoriasis and photosensitivity or psoriasis with atypical morphology.

Suspected lupus

Depending on how strictly the criteria of the American Rheumatism Association are applied (83), two patients (cases 2 and 3, Table X) in whom lupus was suspected could be considered to have SLE, and two other patients (cases 1 and 5) could be considered to have subacute cutaneous LE. From the clinician's standpoint, however, as the courses of these patients evolved, they were initially considered "lupus suspects." The important point is that routine histology in four of these patients was diagnostic of psoriasis, but direct immunofluorescence from the same site was diagnostic (in three) or suggestive (in one) of LE.

Furthermore, each of four photosensitive patients tested had detectable extractable nuclear antigens. Of five photosensitive patients further tested, two also had associated complement deficiencies, one of whom had negative antinuclear antibody results.

The results in these patients exemplify that any patient with psoriasis and photosensitivity in whom no clear cause can be identified require evaluation with routine and direct immunofluorescence biopsy of typical and atypical lesions; testing for

antinuclear antibodies, extractable nuclear antigens, and complement deficiencies; and routine blood tests.

Photosensitive psoriasis

The conditions of only six of our patients (two photosensitive patients without LE, two patients in whom lupus was suspected, one patient with disseminated DLE, and one patient with DLE) could be considered to fulfill the definition of photosensitive psoriasis described by Ros & Eklund (67); they constitute 0.06% of the patients with psoriasis who were seen during the study period, far fewer than the 5.5% Ros & Eklund found. Thirty-four percent of their patients had "experienced sudden onset of light sensitivity, and 26% had been sunbathing intensively at onset" (67). In addition to skin type I and advanced age, they also found that psoriasis affecting the hands and heredity for photosensitivity were more common in photosensitive patients with psoriasis. Several of our patients clearly had "psoriasis" affecting the hands, and heredity for LE and connective tissue diseases is well established (98). Thus, one wonders whether occult LE may account for the photosensitivity in some of the patients described by Ros & Eklund (67). It would be helpful to know whether the Hep-2 antinuclear antibodies, other extractable nuclear antigens, and direct immunofluorescence studies were also negative in their patients.

Treatment

In these patients, only general comments can be made regarding treatment, which must be individualized (99). Obviously, sun protection, sun avoidance, and elimination of offending agents are required for appropriate patients.

For patients with PMLE and photosensitive patients who have psoriasis without LE or other identifiable causes, PUVA or UVB may be the most helpful (69, 100). Prophylactic β-carotene has been suggested but was not found useful in a controlled study (101). Antimalarials may also help (102) and were particularly effective in the patients described by Bielicky & Kvícalová (64), although their potential deleterious effects on psoriasis must be considered.

UVL therapy (B, Goeckerman, or PUVA) may worsen LE. Among patients with psoriasis who have LE, UVL should be considered for treatment of psoriasis when LE is inactive and only when other treatments are ineffective. UVL as part of the Goeckerman regimen may be effective if given carefully by avoiding erythema.

Cytotoxic agents were frequently helpful for psoriasis without aggravating LE, although side effects were frequent. Methotrexate was helpful for several patients in the series of Lynch & Roenigk (23). Retinoids were also beneficial for most patients with psoriasis without aggravating LE. Etretinate was helpful for control of both diseases in two patients described by Green et al. (12) who were intolerant of other medications. Azathioprine was helpful for two severely affected patients who were unresponsive to other regimens (103).

SUMMARY

The coexistence of psoriasis with LE or other photosensitive disorders is rare in our patient population, occurring in 0.69% of patients with psoriasis and 1.1% of those with LE.

PMLE was the most common cause of photosensitivity in psoriatic patients without LE, occurring in 32%. Less common causes included drug-related photosensitivity (thiazides and thiazide derivatives in four of the five cases), PUVA reactions, and photocontact reactions. The Goeckerman regimen or UVB applied in a cautious, well-controlled atmosphere was generally well tolerated in this group, including patients with PMLE.

Photosensitivity occurred in 50% of our patients with psoriasis and LE, and it was secondary to LE in 70% of cases. Most patients were female and had SLE. Psoriasis developed first in 55% of the cases. Studies that were useful for distinguishing photosensitive from nonphotosensitive patients with SLE included determination of antibodies to extractable nuclear antigens (67% versus 14%), double-stranded DNA (64% versus 9%), and skin biopsy for direct immunofluorescence (58% versus 27%).

Occasional patients have features suggestive of photosensitivity with or without signs or symptoms of LE. These patients may have atypical psoriatic plaques occasionally yielding routine histology diagnostic of psoriasis with direct immunofluorescence results suggestive of lupus. Frequently, connective tissue serology findings are positive, and affected patients require close follow-up for the development of LE.

In patients with psoriasis and suspected photosensitivity, we recommend a careful history and examination, skin biopsy for routine histology and direct immunofluorescence, blood tests including determination of antibodies to antinuclear antibodies (Hep-2 substrate if negative on routine substrate), extractable nuclear antigens, and double-stranded DNA, and phototesting when indicated. Large-scale prospective studies are required before the most appropriate therapy for patients with psoriasis and LE can be recommended.

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