Am J Clin Dermatol 2002; 3 (4): 239-246
1175-0561/02/0004-0239/$25.00/0

Photosensitivity Disorders
Cause, Effect and Management

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Contents

Abstract

Abnormal photosensitivity syndromes form a significant and common group of skin diseases. They include primary (idiopathic) photodermatoses such as polymorphic light eruption (PLE), chronic actinic dermatitis (CAD), actinic prurigo, hydroa vacciniforme and solar urticaria, in addition to drug- and chemical-induced photosensitivity and photo-exacerbated dermatoses. They can be extremely disabling and difficult to diagnose.

PLE, characterized by a recurrent pruritic papulo-vesicular eruption of affected skin within hours of sun exposure, is best managed by restriction of ultraviolet radiation (UVR) exposure and the use of high sun protection factor (SPF) sunscreens. If these measures are insufficient, prophylactic phototherapy with PUVA, broadband UVB or narrowband UVB (TL-01) for several weeks during spring may be necessary. CAD manifests as a dermatitis of chronically sun-exposed skin. Again, UVR exposure needs to be restricted; cyclosporine, azathioprine or PUVA may also be necessary. Actinic prurigo is characterized by the presence of excoriated papules and nodules on the face and limbs, most prominent and numerous distally. Actinic prurigo is managed again by restriction of UVR and the use of high SPF sunscreens; PUVA or broadband UVB therapy, or low doses of thalidomide may be necessary. Hydroa vacciniforme causes crops of discrete erythematous macules, 2 to 3mm in size, that evolve into blisters within a couple of days of sun exposure. Treatment for this rare disease is difficult; absorbent sunscreens and restricted UVR exposure may help. Solar urticaria is characterized by acute erythema and urticarial wealing after exposure to UVR. Treatment options for solar urticaria include non-sedating antihistamines such as fexofenadine and cetirizine; other options include absorbent sunscreens, restriction of UVR at the relevant wavelength, maintenance of a non-responsive state with natural or artificial light exposure and plasmapheresis.

Industrial, cosmetic and therapeutic agents can induce exogenous drug- or chemical-induced photosensitiv-

ity. The clinical pattern is highly varied, depending on the agent; treatment is based on removal of the photosensitizer along with restriction of UVR exposure.

Predominantly non-photosensitive dermatoses may also be exacerbated or precipitated by UVR; exposure to UVR should be reduced and sunscreens should be advocated, along with appropriate treatment of the underlying disease.

1. Ultraviolet and Visible Radiation

Radiation is a form of energy defined, by its wavelength, which may be absorbed by specific molecules with appropriate structural conformations. Ultraviolet radiation (UVR) and visible light are part of the spectrum of electromagnetic radiation (EMR) emitted by the sun and various artificial sources. This radiation leads to an increase in molecular energy in suitable circumstances, and may stimulate chemical changes resulting in an observable clinical effect such as skin photoaging and cancer or a photosensitivity syndrome (photodermatosis). For the expression of such changes, the skin must contain an absorber of UVR or visible light (a photosensitizer) which initiates the chain of events leading to the abnormal cutaneous response.

Visible radiation and UVR make up only a very small part of the total EMR spectrum; UVR spans wavelengths of 100 to 400nm and visible light wavelengths of 400 to 800nm.[1] Sunlight that reaches the surface of the earth (terrestrial sunlight) includes all wavelengths above 290nm, as well as a large amount of infrared radiation which produces heat. UVR is further subdivided into three smaller wavebands on the basis of differing biological effects. Thus, ultraviolet C (100 to 280nm) is not present in terrestrial sunlight, or in the emissions from most publicly available artificial sources, and will not be discussed in this review. However, ultraviolet B [(UVB); 280 to 315nm] is very active in human skin, and exposure to it readily induces sunburn, tanning, and many of the photodermatoses. Skin cancers and ageing may occur following chronic repeated exposure. UVB is also present in radiation from such artificial sources as sunlamps and arc welding equipment. Ultraviolet A (UVA), comprising UVA-1 (340 to 400nm) and UVA-2 (315 to 340nm), is a much less active waveband in human skin although it can still produce sunburn and tanning at doses approximately1000-fold greater than that needed with UVB. UVA irradiation is thought to be responsible for many photodermatoses, and chronic exposure may lead to degenerative changes within the skin. In addition to sunlight, UVA is present in emissions from sunbeds and psoralen photochemotherapy [psoralens and ultraviolet A (PUVA)] lamps. Unlike UVB, UVA can be transmitted through window glass; patients who are severely affected by UVA-induced photodermatoses may therefore need additional screening films applied to car and house windows.

2. Primary Photodermatoses

2.1 Polymorphic Light Eruption

Polymorphic light eruption (PLE) is the most common of the photodermatoses,[2] popularly termed 'sun poisoning'. It has a prevalence of 10% in temperate climates, usually affecting young women, with females predominating by about 3 : 1.[3] The cause of PLE is not known, although a genetically-determined basis has been demonstrated, as reflected in the much higher concordance in monozygotic versus dizygotic twins.[4] Histologically, PLE is characterized by a marked perivascular lymphocytic dermal infiltrate. Exposure to sunlight or to artificial sources of light can induce the reaction. The active wavelengths appear to be UVB in many cases, although UVA and occasionally visible light may sometimes contribute to the skin reaction. In temperate regions, the eruption usually recurs with exposure to early summer sun, although winter sunshine enhanced by reflection from snow may occasionally be sufficient.

2.1.1 Clinical Appearance

All racial groups can be affected by PLE, characterized by a recurrent itchy erythematous papular eruption affecting some or all exposed skin within hours of sufficient sun exposure (figure 1); the rash lasts from hours to several days before fading completely in the absence of further exposure. The lesions may be myriad, pinhead-sized, confluent and often whitish or yellowish on an erythematous background, or discrete 2 to 3mm papules clustered in groups. Occasionally vesicles, plaques or generalized erythema and swelling without papules may occur, the last particularly on the face. PLE characteristically affects the nose, malar areas of the cheeks and chin, sides and back of the neck, upper chest, backs of the hands and dorsolateral aspects of the arms. Some clothing, particularly if loose-weave or close-fitting, can allow the passage of radiation to cause a rash on covered sites. The eruption nearly always occurs symmetrically, provided both sides of the body have been exposed. Frequently exposed areas of the body may become tolerant to UVR, especially the habitually uncovered areas such as the face or backs of the hands. In some people, especially those affected only after unaccustomed sun exposure such as on holiday, 2 days or more initial exposure may be necessary before the rash appears.

2.1.2 Diagnosis

The history of eruption in PLE is sufficient to enable a diagnosis supported, if present, by the clinical appearance of the rash. However, titers of antinuclear antibodies and antibodies to extractable nuclear antigens should always be measured to exclude lupus erythematosus, while porphyrin testing should be undertaken to exclude erythropoietic protoporphyria. Irradiation skin tests with the monochromator or broadband UVB sources often induce abnormal reactions, though not usually a rash itself, and may indicate the action spectrum for the rash.

2.1.3 Differential Diagnosis

In some patients, often adolescents, classical PLE may coexist with actinic prurigo which is probably a persistent form of PLE characterized by excoriated papules of the exposed areas. PLE must be distinguished from solar urticaria which is characterized by whealing and usually occurring within 5 minutes of sun exposure and persisting for only an hour after covering up. In erythropoietic protoporphyria, skin pain alone is characteristic following UVR exposure, and red blood cell protoporphyrin levels are abnormal. Erythema multiforme may occur after sun exposure with the same time course and on the same sites as PLE, but histological examination will distinguish the two conditions. UVR-exacerbated atopic eczema also has a similar course to PLE but may be distinguished by its morphology and other evidence of atopy in the patient.

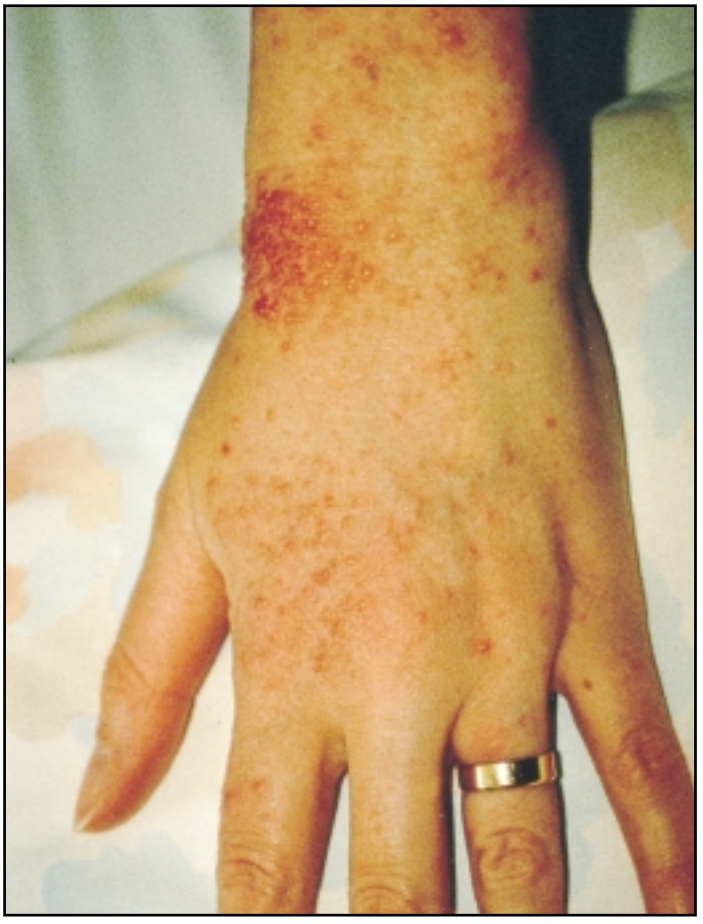

Fig. 1. Polymorphic light eruption.

2.1.4 Management

Restriction of UVR exposure and the use of high sun protection factor (SPF) sunscreens (with efficacy against both UVB and UVA) are important first-line therapies. PUVA, broadband UVB and narrowband UVB with 311nm lamps (TL-01) are also usually prophylactically effective in those patients able to travel to an irradiation centre for treatment.[5] Low-dose irradiation is necessary two to three times weekly for several weeks each spring or before holidays in the sun. TL-01 is considered as effective as PUVA where these treatments have been compared,[6] and thus may well be the treatment of choice in the future, since it has the advantage of being simpler to administer. PLE generally persists or recurs in the absence of treatment.

2.2 Chronic Actinic Dermatitis

The term chronic actinic dermatitis[7] encapsulates several previously-reported forms of photodermatitis. These include the severe variant, actinic reticuloid (the first form of the disease to be described),[8] the milder form of photosensitive eczema[9] and various combinations of the two, including photosensitivity dermatitis and actinic reticuloid syndrome.[10] Chronic actinic dermatitis is relatively common, affecting 1 : 6000 patients in the Tayside region of the UK, usually middle-aged and elderly males (table I), although up to 22% of patients are women.[11] Although the cause of chronic actinic dermatitis is unknown, it appears likely to be a delayed-type hypersensitivity reaction against an endogenous cutaneous photo-induced allergen, leading to precisely the same features as allergic contact dermatitis. However, if a specific photosensitizer is incriminated as the cause of an apparent case of chronic actinic dermatitis, the condition should then be described as a contact photodermatitis (see section 3). Photosensitizers which cause this must be distinguished from incidental photosensitizers, merely exacerbating the clinical picture of chronic actinic dermatitis. Sunscreens in particular may be implicated in this process.

2.2.1 Role of Specific Allergens

Airborne allergic contact dermatitis may mimic the clinical picture of chronic actinic dermatitis, but more than this, it appears likely that there is also a causal relationship between this form of contact sensitivity and the development of chronic actinic derma-

titis. In particular, contact sensitivity to oleoresins (such as sesquiterpene lactone) from plants of the Compositae family, especially chrysanthemums, has been implicated and perhaps to a lesser extent, phosphorus sesquisulphide, colophony, rubber, metals, and allergens used in medicaments, perfumes and sunscreens may also be involved. The causative mechanism of these agents in inducing the photosensitivity of chronic actinic dermatitis remains unknown. It is conceivable that chronically eczematous skin, such as that in chronic actinic dermatitis, may enable the easier penetration of oleoresins and other airborne antigens leading to a secondary and incidental contact dermatitis merely exacerbating and not causing the chronic actinic dermatitis.

A large number of cases of chronic actinic dermatitis are associated with positive patch test results, but in a few cases, a positive photopatch test reveals chronic actinic dermatitis to be a photocontact dermatitis or more rarely a drug photosensitivity. Some patients in fact are cases of photocontact dermatitis to musk ambrette, although this has now largely been removed from male toilet preparations. The remainder of the rare positive photopatch-test results show only incidental photosensitizers which may aggravate but do not cause the disease.

2.2.2 Clinical Appearance

Both Black- and White-skinned people may be affected by chronic actinic dermatitis, frequently with a preceding history of other endogenous or exogenous eczema. Chronic actinic dermatitis is worse in summer and following sun exposure, although this relationship is not always reported by patients. Widespread chronic eczematous changes, comprising of scaly lichenification or infiltrated plaques, occur particularly on the exposed skin of the face, scalp, back and sides of the neck, upper chest, and the dorsal surfaces of the arms and backs of the hands (figure 2). Islands of exposed skin may sometimes be unaffected while large areas of covered skin may instead be affected, usually with patchy eczematous changes or sometimes with confluent erythema. Infiltration of the skin leads to an accentuation of skin markings on the face and a rare tendency to a leonine facies in severe cases. Sparing in the depth of skin creases and skin folds may occur, as well as finger-webs and upper eyelids. Palmar and plantar eczema are not unusual. Hair, especially on the eyebrows and eyelashes may be short and broken off or lost, presumably from scratching, while large areas of marked hyper- or hypopigmentation are noted in the exposed or covered areas of the skin. Generalized erythroderma may develop in severe cases, usually with more marked changes on the exposed areas. In very rare cases, however, the erythroderma is uniform with no accentuation on exposed sites. Such patients are usually very light sensitive. Chronic actinic dermatitis must therefore be remembered as a possible cause of generalized erythroderma. Gradual spontaneous remission of the condition may sometimes occur.

Table I. Factors predisposing to or associated with chronic actinic dermatitis[10]

Male gender
Increasing age
Outdoor activities
Atopic eczema
HIV infection
Allergic contact dermatitis

2.2.3 Diagnosis

Irradiation skin tests are an essential investigation tool in chronic actinic dermatitis, both to establish the diagnosis and to determine the wavelengths causing the condition in order to optimize logical treatment. The major criteria for the diagnosis of chronic actinic dermatitis are listed in table II. Lower doses of radiation, than that used in patients without chronic actinic dermatitis, produce erythema and frequently marked swelling of irradiated sites in the UVB range, and often in the UVA and occasionally visible light ranges as well. Very rarely the irradiation tests are normal for a short period after onset of the disease, and may need to be repeated some weeks to months later if there is a strong clinical suggestion of chronic actinic dermatitis. A skin biopsy shows eczematous changes usually with a fairly marked deep dermal lymphohistiocytic infiltrate or, especially in sections from infiltrated plaques, epidermotropism and Pautrier-like nests of cells strongly reminiscent of cutaneous T cell lymphoma (CTCL). Patch tests are necessary to exclude airborne contact dermatitis as a cause of the rash if light sensitivity is shown not to be present, and to reveal contact sensitizers, particularly to sunscreen constituents, which may be exacerbating the chronic actinic dermatitis.

2.2.4 Differential Diagnosis

Chronic actinic dermatitis must be differentiated from simple airborne contact dermatitis; patients with infiltrated plaques may instead have CTCL, particularly since the histology of such cases of chronic actinic dermatitis and of CTCL may be difficult to distinguish. The severe light sensitivity of chronic actinic dermatitis is the distinguishing factor, although CTCL may occasionally be associated with mild light sensitivity. The erythroderma of chronic actinic dermatitis may resemble that of the Sézary syndrome, particularly since large numbers of circulating Sézary cells may be present in chronic actinic dermatitis, but the marked light sensitivity in chronic actinic dermatitis again serves to distinguish the condition.

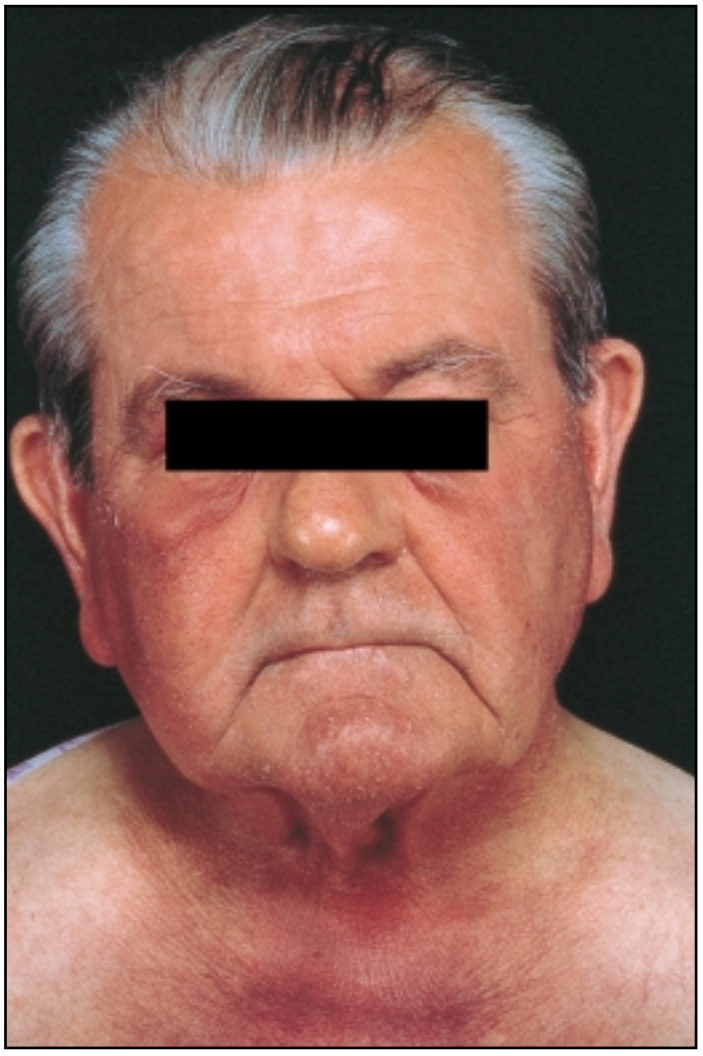

Fig. 2. Chronic actinic dermatitis.

2.2.5 Management

The first step in the management of chronic actinic dermatitis is restriction of UVR exposure (and if necessary to visible light), and in severe cases patients may need to be confined to relatively dark conditions. Improved lighting conditions are possible with the use of commercially available plastic film blinds which strongly filter radiation below specified wavelengths in the long UVA or short visible ranges. Sunscreens should also be used but are most likely to be effective in cases of chronic actinic dermatitis sensitive to UVB alone, although UVA screens are now also very effective.

The oral administration of azathioprine seems moderately effective in about two-thirds of the patients with chronic actinic dermatitis and may be used in refractory cases; 1.0 to 2.5 mg/kg/day usually helps after about a month or so of therapy and some patients then remain free of the disease. Cyclosporine, at doses of 3.5 to 5.0 mg/kg/day may also be beneficial. PUVA therapy given three to five times weekly for several months under gradually reducing oral steroid-cover, can also be effective. Very low doses (0.25 J/cm^2 or less) of UVA are initially necessary to avoid exacerbating the disease, but the dose can be increased gradually as immunological tolerance develops and the disease begins to remit. Some cases remit spontaneously; most others respond to azathioprine, cyclosporine or PUVA. A minority of patients still remain intractable and socially disabled, as in the past, before relatively effective treatment became available.

2.3 Actinic Prurigo

Actinic prurigo is characterized by the presence of excoriated, often crusted and scabbed papules and occasionally nodules, on the face and limbs in particular, most prominent and numerous distally. Very shallow linear, flat or punctate scars may occur on the face. The condition worsens in summer and after sun exposure, though this relationship is often not evident to the patient. Actinic prurigo generally affects children and adolescents, apparently resolving during puberty in most cases, although it may develop or persist into adulthood.[2] Adult native North and South Americans in particular appear to be regularly affected. Although of unknown cause, tissue typing of patients with AP has demonstrated a prevalence of human leukocyte antigen–D related B1*04 (HLA-DRB1*04) of 90% in Caucasians (normally 30%), and this may suggest an abnormal response to an UV-induced peptide antigen. In about two-thirds of patients, the action spectrum for erythema has been reported as abnormal, more commonly to UVA than UVB.

Actinic prurigo may resemble atopic eczema, insect bites, prurigo nodularis or, if scarring is severe, erythropoietic protoporphyria. It is improved by the restriction of UVR exposure and the use of high-SPF sunscreens. PUVA or broadband UVB therapy given as for PLE may be helpful. In patients who are otherwise nonresponsive, however, intermittent courses of low dose thalidomide at doses of 50 to 100mg at night is also effective. However, its teratogenicity and tendency to induce a peripheral neuropathy have severely restricted its use. One option may be to clear the eruption with thalidomide and maintain the clearance with low dose phototherapy.

2.4 Hydroa Vacciniforme

Hydroa vacciniforme is a very rare disease occurring almost exclusively in children although, very rarely, the middle-aged or even elderly may be affected.[2] Although the cause is unknown, an association with HLA DRB1*04 has recently been claimed.[12] Recurrent crops of discrete 2 to 3mm sized erythematous macules

evolve into blisters hours to a day or two after sun exposure in summer. Healing occurs within days with umbilification followed by crusting and unsightly, pitted, varioliform scarring. The face and backs of the hands are most frequently affected. Diagnosis is made on clinical and histological grounds; biopsy of a blister site shows mid-epidermal necrosis and a lymphocytic infiltrate. Phototesting with UVA may also trigger lesions. Absorbent sunscreens and the restriction of UVR exposure help somewhat but no treatment has been universally successful. Chloroquine and PUVA have been advocated, but they seem usually to be unhelpful, while thalidomide and cyclosporine are also of unknown efficacy.

2.5 Solar Urticaria

New cases of solar urticaria have been described in age-groups ranging from one year to the eighth decade.[13] Solar urticaria appears to have an immunological basis[14] and any part of the UVR or visible light spectrum may induce the condition, but the evoking spectrum wavelengths are generally specific and consistent for a given patient. Five to 10 minutes after exposure, tingling irritation occurs over the exposed areas followed rapidly by erythema and whealing. The wheals frequently become confluent with a well defined ridge of skin at the margin of the exposed sites. Habitually exposed areas such as the face and backs of hands may not be affected. The eruption settles completely within 1 to 2 hours of cessation of exposure. Repeated exposures up to 24 hours apart sometimes lead to temporary loss of reactivity.

Table II. Diagnosis of chronic actinic dermatitis[11]

Clinical features	Mainly eczematous eruption of exposed areas
Histology (not a prerequisite for diagnosis)	Chronic eczema, with or without CTCL-like changes
Phototests (essential for diagnosis)	Irradiation monochromator: reduction in 24-hour MED and exaggerated papular responses to UVB, usually UVA and rarely visible wavelengths
	Broadband source: reduction in the 24-hour MED; induction of eczema is also possible
Patch and photopatch tests (essential ancillary tests)	Abnormalities frequently detected, often to ubiquitous airborne allergens or topical medications
Porphyrins	Normal
Serology	Negative for DNA, Ro and La antibodies

CTCL = cutaneous T cell lymphoma; **MED** = minimal erythemal dose; **UVA** = ultraviolet A; **UVB** = ultraviolet B.

Solar urticaria may be confused with PLE because of an apparently similar time course, but solar urticaria evolves and resolves much more rapidly. Confusion may similarly arise with other rapidly exacerbated or induced photodermatoses such as lupus erythematosus and nonurticarial photosensitivity to topical agents. In addition, solar urticaria may occasionally be associated with PLE or systemic lupus erythematosus. It may very rarely be a feature of the porphyrias, particularly erythropoietic protoporphyria, or a manifestation of photosensitivity to topically applied agents, notably some coal tar compounds and dyes, or oral drugs such as benoxaprofen, now withdrawn.

A definitive diagnosis in doubtful cases is best made by the examination of lesions induced by sunlight or artificial sources; the latter, however, are not always effective at inducing lesions and failure to do so does not necessarily invalidate the diagnosis. Monochromatic irradiation should be used if possible to determine the causative wavelengths prior to initiation of treatment. The relatively rare UVB-induced solar urticaria may be treated with high SPF absorbent sunscreens and restriction of UVB exposure, while nonsedating histamine H_1 receptor antagonists including fexofenadine and cetirizine are effective in about half of the cases, if taken at sufficient doses. Maintenance of a nonresponsive state by recurrent exposure to sunlight or artificial lamps may help. PUVA and TL-01 have been advocated but are not always effective. Solar urticaria remains difficult to treat; in intractable cases, however, plasmapheresis is occasionally used to eliminate the putative photoallergen from the plasma. Reasonable results have been observed, some patients achieving remission for more than 1 year.

3. Drug and Chemical Photosensitivity

Exogenous drug- or chemical-induced photosensitivity is induced by three main groups of agents; industrial, cosmetic and therapeutic. Drug-induced photosensitivity usually manifests as a phototoxic response (usually a sunburn-like reaction) to both oral and topical agents (table III) or, less commonly, a photoallergic (eczematous) response.[15] Clinical photosensitizers generally absorb UVA, the ensuing photochemical reactions then leading to a variety of inflammatory or immunologically mediated clinical features depending on where the photosensitizer is principally located within the skin. Only certain compounds are structurally able to absorb radiation to produce these effects; normally such compounds are organic, planar, cyclic, polycyclic or long-chain molecules with a series of alternating single and double chemical bonds.

The clinical pattern of drug photosensitivity is highly variable, with several main patterns of reaction; such reactions occur

Table III. Patterns of cutaneous phototoxicity[13]

Skin reactions	Photosensitizers
Exaggerated sunburn	Fluoroquinolone antibiotics, chlorpromazine, amiodarone, thiazides, quinine, tetracyclines
Prickling; immediate erythema; edema or urticaria with higher doses	Coal tar, pitch, anthraquinone-based dyestuffs, amiodarone, chlorpromazine
Late-onset erythema; blisters with higher doses	Psoralens, phytophotodermatitis, berloque dermatitis
Increased skin fragility with blisters from trauma (pseudoporphyria)	Nalidixic acid, furosemide (frusemide), tetracyclines, naproxen, amiodarone

only on sites exposed to both photosensitizer and UVR (figure 3), although photoallergic reactions may spread. Thus contact phototoxic agents such as tar, pitch, dyes, psoralens and certain systemic sensitizers such as the tetracycline antibiotics and the endogenous porphyrins of erythropoietic protoporphyria may induce immediate smarting of affected areas of skin, often associated with the later development of erythema, edema, blistering and sometimes whealing. On other occasions, these latter signs may predominate without the clear occurrence of any immediate or early reaction. Other systemic phototoxic agents such as furosemide (frusemide) and nalidixic acid and the endogenous porphyrins of porphyria cutanea tarda, variegate porphyria and hereditary coproporphyria, may induce skin fragility with subepidermal bulla formation on areas exposed to light, along with crusting, scarring and milia formation. Some contact photosensitizers induce an eczematous reaction at affected sites; this may become chronic and eventually progress to chronic actinic dermatitis, with the photosensitizer no longer needing to be present for the reaction to continue. Musk ambrette and 6-methylcoumarin used as fragrances and fixatives in cosmetics have typically induced this response in the past.

In the diagnosis of drug photosensitivity, irradiation skin tests with a monochromator or other artificial sources may be normal or elicit smarting, erythema, whealing or papular responses at lower than normal UVA doses, if the photosensitizer is present in the exposed skin. There may be UVB or visible light abnormalities as well and, occasionally it may be difficult to distinguish these phototest abnormalities from those of chronic actinic dermatitis; they are usually more exaggerated and broad-spectrum in the latter. Photopatch tests with the suspected photosensitizer should be undertaken and may sometimes be positive, but negative results do not exclude photosensitivity, in that the agent used may not penetrate to the appropriate cutaneous site.

The first-line of treatment is removal of the photosensitizer while restriction of UVR exposure is needed until this is done. Sunscreens are usually only moderately helpful since photosensitivity is generally induced by UVA wavelengths. Recent onset photosensitivity to topical agents usually starts to resolve immediately after the photosensitizer is removed and as quickly as the skin can heal. Resolution following the removal of an oral agent may sometimes take longer, presumably until all body stores of the drug have been metabolized or excreted.

4. Photoexacerbated Dermatoses

Predominantly nonphotosensitive dermatoses may be precipitated or exacerbated by UVR (table IV) in some patients, while in other patients no effect or even improvement may occur. The mechanisms are unknown, but may involve an accentuation of the basic immunological disease response in some patients or an additive inflammatory response in others; UVB appears usu-

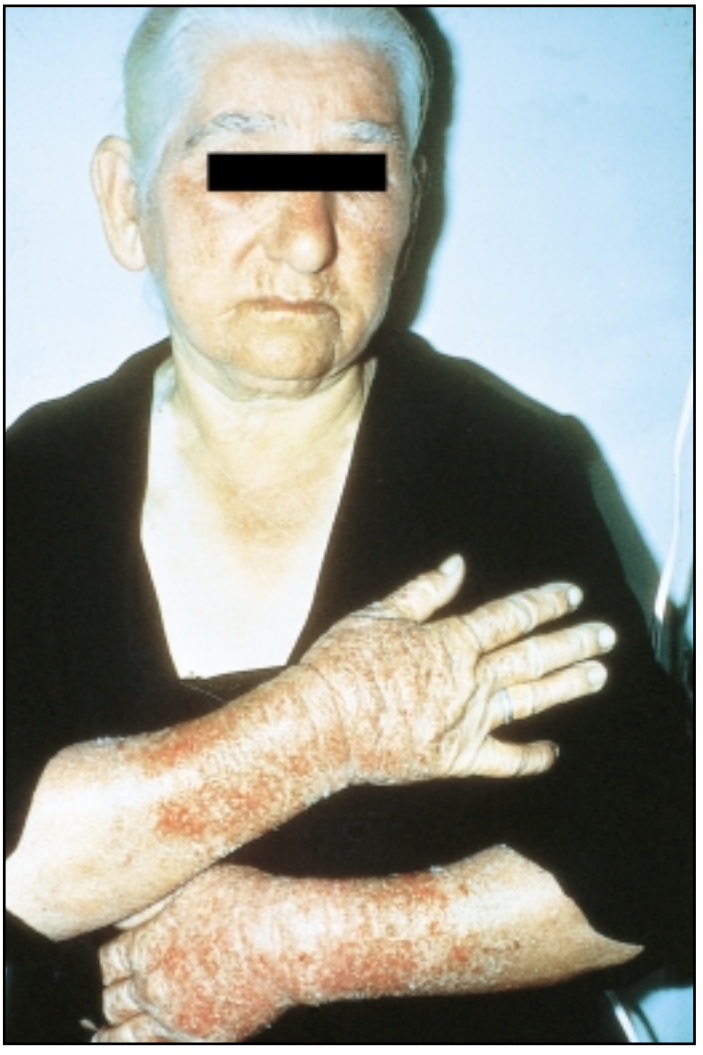

Fig. 3. Exogenous drug photosensitivity.

Table IV. Ultraviolet radiation (UVR)-exacerbated dermatoses

Rosacea
Atopic eczema[a]
Erythema multiforme
Psoriasis[a]
Herpes simplex
Lupus erythematosus (discoid, subacute and systemic)
Dermatomyositis
Lichen planus
Seborrheic dermatitis[a]
Darier's disease
Pemphigus foliaceus
Bullous pemphigoid
Cutaneous T cell lymphoma[a]

a May be improved instead by UVR in many patients.

ally to be responsible. The dermatosis in question may be aggravated on all exposed areas, although not all exposed areas are necessarily affected. Usually the diagnosis is relatively clear from the clinical features of the underlying dermatosis and the history of sun-related exacerbation. However, transient conditions such as erythema multiforme and mild atopic eczema may be confused with PLE and seborrheic eczema with chronic actinic dermatitis. Erythematous disorders such a lupus erythematosus may also be confused with erythropoietic protoporphyria, solar urticaria or exogenous drug and chemical-induced photosensitivity. Skin irradiation tests with artificial light sources and other investigations are generally normal in these light-exacerbated dermatoses. UVB exposure should be restricted and sunscreens used along with treatment of the underlying dermatosis; the light sensitivity may dramatically disappear with the last measure.

5. Conclusion

Abnormal photosensitivity syndromes form a significant group of skin diseases; they can be extremely disabling and difficult to diagnose and treat. Both abnormal skin reactivity and exposure of the skin to the appropriate wavelengths of UVR or visible light are necessary for the expression of these diseases, many of which can now be effectively treated once the correct diagnosis has been made.

Acknowledgments

The authors declare no conflict of interest. Dr Millard is funded by a research grant from the Special Trustees of St Thomas' Hospital.

References

1. Diffey BL. Human exposure to ultraviolet radiation. In: Hawk JLM, editor. Photodermatology. London: Arnold, 1999: 5-24
2. Norris PG, Hawk JLM. The idiopathic photodermatoses: polymorphic light eruption, actinic prurigo and hydroa vacciniforme. In: Hawk JLM, editor. Photodermatology. London: Arnold, 1999: 103-12
3. Pao C, Norris PG, Corbett M, et al. Polymorphic light eruption: prevalence in Australia and England. Br J Dermatol 1994; 130: 62-4
4. Millard TP, Bataille V, Snieder H, et al. The heritability of polymorphic light eruption. J Invest Dermatol 2000; 115: 467-70
5. Millard TP, Hawk JLM, Travis LB, et al. Treatment of polymorphic light eruption. J Dermatol Treat 2000; 11: 195-9
6. Bilsland D, George SA, Gibbs NK, et al. A comparison of narrow band phototherapy (TL-01) and photochemotherapy (PUVA) in the management of polymorphic light eruption. Br J Dermatol 1993; 129 (6): 708-12
7. Hawk JLM, Magnus IA. Chronic actinic dermatitis: an idiopathic photosensitivity syndrome including actinic reticuloid and photosensitive eczema. Br J Dermatol 1979; 101 Suppl. 17: 24
8. Ive FA, Magnus IA, Warin RP, et al. Actinic reticuloid; a chronic dermatosis associated with severe photosensitivity and the histological resemblance to lymphoma. Br J Dermatol 1969; 81: 469-85
9. Ramsay CA, Kobza-Black A. Photosensitive eczema. Trans St. Johns Hosp Dermatol Soc 1973; 59: 152-8
10. Frain-Bell W, Lakshmipathi T, Rogers J, et al. The syndrome of chronic photosensitivity dermatitis and actinic reticuloid. Br J Dermatol 1974; 91: 617-34
11. Menagé H, Hawk JLM. The idiopathic photodermatoses: chronic actinic dermatitis. In: Hawk JLM, editor. Photodermatology. London: Arnold, 1999: 127-210
12. Grabczynska SA, McGregor JM, Kondeatis E, et al. Actinic prurigo and polymorphic light eruption: common pathogenesis and the importance of HLA-DR4/DRB1*0407. Br J Dermatol 1999; 140 (2): 232-6
13. Ferguson J. Idiopathic solar urticaria: natural history and response to non-sedative antihistamine therapy, a study of 26 cases. Br J Dermatol 1988; 119 Suppl. 33: 16
14. Hölzle E. The idiopathic photodermatoses: solar urticaria. In: Hawk JLM, editor. Photodermatology. London: Arnold, 1999: 113-26
15. Ferguson J. Drug and chemical photosensitivity. In: Hawk JLM, editor. Photodermatology. London: Arnold, 1999: 155-70

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