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Photosensitive disorders of the skin with ocular involvement☆

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Abstract Patients with photosensitive disorders of the skin may present with ocular manifestations that are evident at birth or may be manifested later with progression of the disorder. Dermatologists should be able to recognize these and appropriately refer patients for further management. Ocular involvement associated with immunologically mediated photodermatoses, drug- and chemical-induced photosensitivity, photodermatoses associated with defective DNA repair/chromosome instability, and photoaggravated dermatoses are reviewed.

Photodermatoses are commonly classified into four general groups: (1) immunologically mediated photodermatoses; (2) drug- and chemical-induced photosensitivity; (3) photodermatoses associated with defective DNA repair/chromosome instability; and (4) photoaggravated dermatoses. Photodermatoses in these groups with ocular involvement will be discussed. In addition, skin diseases associated with photophobia are also described.

Immunologically mediated photodermatoses

Actinic prurigo (AP)

Actinic prurigo is a pruritic, erythematous, papular or nodular eruption induced by sunlight that occurs mainly, but not exclusively, on sun-exposed skin. It has been described as a variant of polymorphic light eruption (PMLE). Actinic prurigo usually presents in childhood and can resolve during adolescence. It has been reported in all races; however, a higher prevalence has been described in mestizos (mixed European and Native American background). It is also more common in the United Kingdom. A strong association with HLA-DR4 (DRB1*0407) has been observed in patients from the United Kingdom and in mestizo patients, suggesting a genetic susceptibility.[1,2] Erythematous papules and nodules appear in sun-exposed areas, including the face and extremities. Lesions may have hemorrhagic crusts or lichenification and can heal with scarring. Cheilitis is a common feature (Figure 1). Ophthalmic findings include conjunctivitis, photophobia, pinguecula/pterygium, hyperemia, Trantas' dots (raised white gelatinous collections), hyaline exudate, and follicles. Pathologic changes of conjunctiva include

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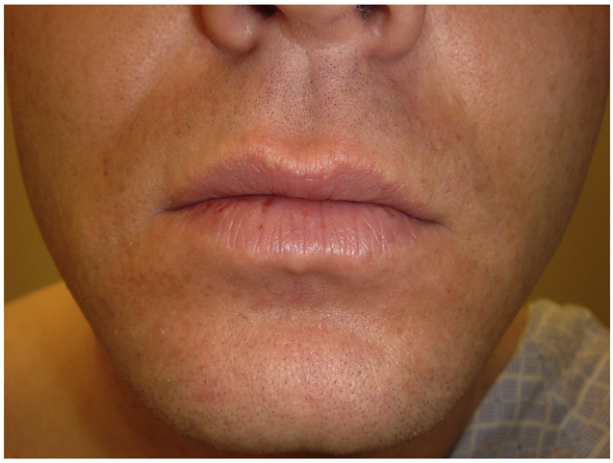

Fig. 1 Cheilitis in a patient with actinic prurigo.

epithelial changes consistent with epidermoid metaplasia, absence of goblet cells, and stromal changes.[3]

AP is provoked by UVR exposure, and although a negative phototesting result does not exclude the diagnosis, phototesting may indicate a decreased minimal erythema dose to UVA, UVB, or both.[4] Mainstays of treatment include strict photoprotection, topical corticosteroids, and topical calcineurin inhibitors for mild disease and NB-UVB, PUVA, or oral thalidomide for more severe disease. Ophthalmic treatments include surgical excision of the conjunctival lesions and treatment with topical cyclosporine eye drops in cases of treatment failure with topical steroids and vitamin A ointments.[5]

Chronic actinic dermatitis

Chronic actinic dermatitis (CAD) is an uncommon, chronic eczematous dermatitis with a predominant photodistribution and, clinically and histologically, lichenification. It is induced by ultraviolet radiation and rarely by visible light. CAD is thought to be a delayed-type hypersensitivity response against photo-induced cutaneous antigens, similar to allergic contact dermatitis. It most commonly occurs in older men and presents as a pruritic, eczematous eruption with lichenification on sun-exposed areas of the body with sharp cutoff at lines of clothing. It may also present as erythroderma. Unlike airborne contact dermatitis, there is sparing of the upper eyelids, nasolabial folds, retroauricular areas, and submental areas (Figure 2); however, in long-standing cases, these protected areas may become involved.

Diagnosis relies on differentiating from other photosensitive disorders such as drug-induced photosensitivity; cutaneous T-cell lymphoma should also be considered. Treatment consists of strict photoprotection, avoidance of contact allergens, and topical corticosteroids and calcineurin inhibitors for mild disease and cyclosporine, azathioprine, mycophenolate mofetil, and low-dose PUVA in combination with oral corticosteroids for more severe disease. Recently, NB-UVB with oral corticosteroids has been reported to be effective.[6]

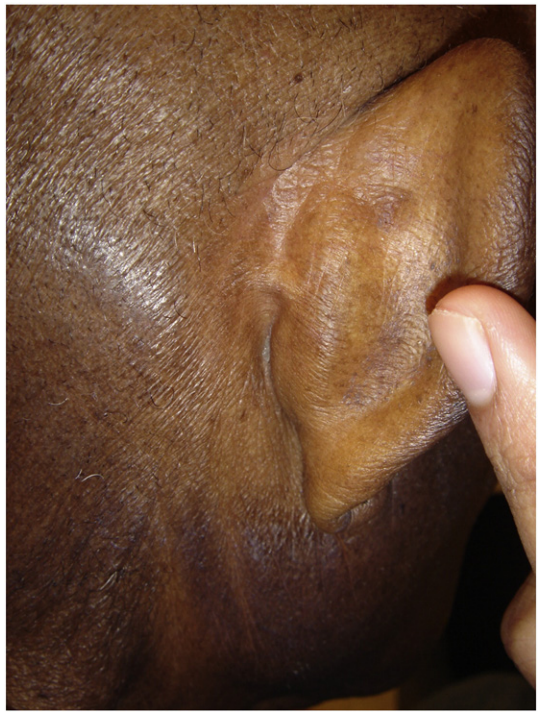

Fig. 2 Sparing of retroauricular area in a patient with chronic actinic dermatitis.

Hydroa vacciniforme

Hydroa vacciniforme (HV) is a rare photodermatosis with an onset in childhood that usually resolves by late teenage years. It presents as clustered, pruritic erythematous macules in exposed skin within hours of exposure to summer sunlight. These lesions often progress to vesicles or bullae that umbilicate and form hemorrhagic crusts, leaving pock-like scars (Figure 3).[7] An association with Epstein-Barr virus has been reported.[8] Ocular findings are less common, although tearing and photophobia can be present. Other ocular findings include cicatricial ectropion, conjunctival chemosis, hyperemia, vesiculation, scarring, and corneal

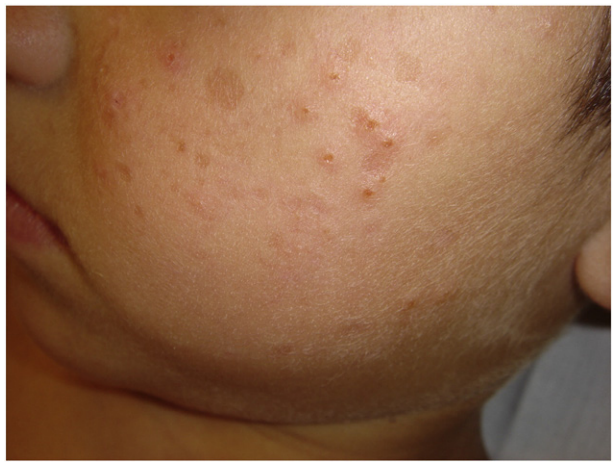

Fig. 3 Pock-like scars in a patient with hydroa vacciniforme.