The springtime eruptions

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The spectrum of idiopathic ultraviolet-induced dermatoses includes the most severe variant, polymorphous light eruption (PMLE), in which erythematous papules, papulovesicles, and plaques form from a delayed type IV hypersensitivity reaction to the sun. Its milder cousins, benign summer light eruption and juvenile springtime eruption, are milder, more transient variants.

Patients may experience sudden-onset, pruritic, sometimes painful papules and papulovesicles or cheilitis within 30 minutes to several hours of exposure to UV light in areas normally covered in the winter months. The rash subsides over 1-7 days (or sooner with effective topical steroid administration and strict sun avoidance) and without scarring. Occasionally, patients experience systemic flulike symptoms after sun exposure. Triggers can be UVA, UVB, or UVC. However, because most cases appear in the spring, describing these variants as benign summer light eruption is something of a misnomer.

These seasonal rashes are often underrecognized in skin of color patients, particularly those with Fitzpatrick skin types III-VI, because many practitioners assume a protective role of melanin (Photochem. Photobiol. Sci. 2013;12:65-77). A study by Kerr and Lim identified 280 patients with photodermatoses; 135 (48%) were African American, 110 (40%) were white, and 35 (12%) were other ethnicities. They noted a significantly higher proportion of African Americans with PMLE, compared with whites (J. Am. Acad. Dermatol. 2007;57:638-43). Also, Native Americans have a hereditary form of PMLE with autosomal dominant inheritance that can involve the face and is most common in patients with Fitzpatrick skin types III-VI.

For sun-sensitive patients, and especially skin of color patients, diagnosis and treatment include ruling out other photosensitive diseases such as systemic lupus, and then counseling about the importance of sun avoidance and the use of sunscreens, which include both UVA and UVB protection. Photophylactic phototherapy or photochemotherapy at the beginning of spring for several weeks may prevent flare-ups throughout the summer. PUVA (psoralen and UVA) therapy, as well as UVB phototherapy, have been successful at preventing flares in several studies. Topical steroids, antihistamines, and oral prednisone are mainstays of treatment for severe flares, alone or in combination with phototherapy. For severe cases, or those recalcitrant to first-line treatment, antimalarials, azathioprine, and thalidomide have been used with variable efficacy.
Particularly at this time of year, I always ask patients with photo-distributed rashes about their ethnicities. One can never assume ethnicity, culture, skin type, background, or even photosensitivity based on skin color alone. I have been surprised by the many patients with dark skin who may have Native American origins who present with photosensitive rashes, or the many patients with hereditary photosensitive rashes with fair skin. Our beautiful, multicultural society makes it harder to define or categorize dermatoses by skin type alone, based on the definitions we have set for skin type in our literature today.

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