Patient with a skin rash

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A fit and healthy 22 year old of Asian ancestry, living in New Zealand with no previous history of asthma or dermatitis, visited the Gold Coast, Queensland, for a vacation. Having been cautioned about the intensity of the Australian sun, he arrived armed with a hat and new SPF130 sunscreen that he had obtained from Japan. On the third day of his vacation he noticed the appearance of many erythematous papules and some vesicles on the dorsum of the forearm, arm and the 'v' of the neck. His face was spared. On the fourth day, the papules coalesced to form swollen, warm, very pruritic, erythematous plaques (Figure 1). The patient commenced taking loratadine 10 mg per day, but did not improve after seven days of treatment.



Figure 1. Erythematous papules coalesce forming swollen, warm plaques

Question 1

What is the importance of the distribution of the lesions?

Question 2

What are the probable causes of this dermatosis?

Question 3

How would you manage this patient?

Answers

Answer 1

The distribution of the lesions on the shoulders and extensor surfaces of the upper limbs and the 'v' of the neck suggest this rash was triggered by exposure to ultraviolet radiation.

Answer 2

There are many types of abnormal reactions to solar radiation. These include:

- photoaggravated diseases such as systemic lupus erythematosus and herpes simplex
- metabolic diseases such as the porphyries, and
- idiopathic reactions such as polymorphous light eruption. This is a common disease characterised by lesions of several types including papules (that coalesce to form plaques), vesicles, or less commonly, hives that appear 1–4 days after expo-

sure and persist for several days. Photoexposed areas are affected especially those protected during the winter (eg. the arms) whereas areas exposed all year (eg. the face) are usually spared. Polymorphous light eruption tends to manifest in spring and summer months, improves with continued sun exposure (a phenomenon referred to as 'hardening') but tends to recur each year and may be more extensive during subsequent summers.'

 photosensitive disorders. These can be divided into two types: phototoxic and photoallergic. Phototoxic reactions can be induced by a variety of agents that produce a rash, not through an allergic mechanism, but by increasing the sensitivity of the skin to ultraviolet radiation. Topical phototoxic agents include psoralens present in plants,

celery and the rind of citrus fruit including bergamot (Citrus bergamia) used as a fixative in the perfume industry. Systemic phototoxic agents such as thiazide diuretics and tetracyclines may cause intense erythema of exposed areas and in severe cases, shedding of the nails (photoonycholysis). In contrast, photoallergic reactions are caused by a different mechanism of damage that involves ultraviolet radiation initiating a reaction between skin proteins and a chemical to form an antigen that produces delayed type hypersensitivity reaction. Topical chemicals associated with photoallergy include halogenated antibacterial and antifungal agents added to soaps and lotions, and cinnamates and oxybenzone found in some sunscreens.2

In establishing the differential diagnosis for this patient, polymorphous light eruption and photoallergic contact dermatitis must be considered. The clinical presentation is highly suggestive of polymorphous light eruption. A photoallergic reaction to the sunscreen is much less probable, not only because it is much rarer than polymorphous light eruption, but also because it would have affected the patient's face, even if he were wearing a hat, due to the substantial reflected sunlight from the water and sand.

Answer 3

Cool compresses plus a high potency topical steroid are usually enough to settle down a polymorphous light eruption. In cases of severe pruritus, chlorpheniramine or doxepine may help. If the patient shows severe discomfort, a short course of prednisone may be necessary.

To prevent recurrences of polymorphous light eruption the patient should be instructed to avoid exposure to the sun at times of maximum intensity (between 11 am and 3 pm). Sunscreens may not be protective, as the condition is caused by longer wavelengths of ultraviolet radiation which may not be filtered out by

many products. Patients who recur with severe disease in spite of these measures may be referred for psoralen ultraviolet A (PUVA) treatment.

References

- 1. Naleway A L. Polymorphous light eruption. Int J Dermatol 2002; 41:377–383.
- Cook N, Freeman S. Report of 19 cases of photoallergic contact dermatitis to sunscreens seen at the Skin and Cancer Foundation. Australas J Dermatol 2001; 42:257–259.