

Solar urticaria confined to areas of bruising

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Summary

Key words:

bruising; ecchymosis; solar urticaria

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Accepted for publication:

31 March 2010

Conflicts of interest:

None declared.

Solar urticaria is a rare immunoglobulin E-mediated photodermatosis characterized by the development of a pathognomic wheal and flare within minutes of photoexposure. A 26-year-old man presented with a history of sun-induced urticaria only in areas of pre-existing bruising, which we were able to replicate with monochromator testing. We were also able to demonstrate, by autologous *in vitro* studies, that a serum factor was responsible.

Solar urticaria is a rare photodermatosis characterized by the development of a wheal and flare usually within minutes of photoexposure. The presumed pathogenesis is that a photoallergen, produced by exposure to ultraviolet (UV) radiation, is recognized by specific immunoglobulin E (IgE) molecules leading to the degranulation of mast cells. We present a case of a patient with a history of sun-induced reactions confined to areas of pre-existing bruising.

Case report

A 26-year-old male patient presented to our department with a 10-year history of sun- and sunbed-induced reactions in areas of pre-existing bruises on his body. He described developing an itchy, red rash occurring within minutes of exposure to sunlight or sunbeds, and clearing within hours. It occurred only on pre-existing sites of bruising and invariably spared normal skin. He described no benefit with anti-histamines.

His haematological, urine and faecal investigations for common photodermatoses were unremarkable including a negative porphyrin screen, negative anti-nuclear, and extractable nuclear antibodies. Examination before testing revealed bruising over the lateral aspect of his left thigh and calf (Fig. 1a).

Monochromator phototesting on normal looking skin elicited no abnormal reactions (Fig. 1b). Testing on bruised skin caused a marked flare with UVB (310 nm) (Fig. 1c) and a less severe but still abnormal reaction with UVA (360 nm) (Fig. 1d). When serum was irradiated at 310 nm *in vitro* and then injected

intradermally, an immediate response was elicited. Autologous non-irradiated serum elicited no abnormal reactions (Fig. 2).

Discussion

In the early 1900s investigators first hypothesized that there was a circulating serum factor that was induced by UV light that went on to cause histamine release. It was Horio and Minami (1) in 1977 who postulated that a precursor substance (chromophore), present in the serum and/or the dermis, could be activated by light and become a photoallergen. A number of potential photoallergens have been discovered over the years using ultrafiltration techniques (2). These serum factors have ranged in size from 25 to 1000 kDa, often correlating with different action spectra, but the definite identification of a responsible photoallergen remains illusive.

Leenutaphong *et al.* (3) in 1989 classified solar urticaria into two IgE-mediated types. Type 1 solar urticaria is caused by an abnormal chromophore present in the serum only in patients with solar urticaria and against which IgE antibodies are directed. These patients would have variably positive passive transfer tests (patient serum injected into dermis of normal patient and then exposed to action spectra) depending on the viable transfer of the chromophore. Type II solar urticaria is caused by abnormal circulating IgE antibodies against a normal chromophore. In this case passive transfer tests would always yield positive results.

There are a number of varied clinical manifestations of solar urticaria in the literature including cases of delayed onset and also

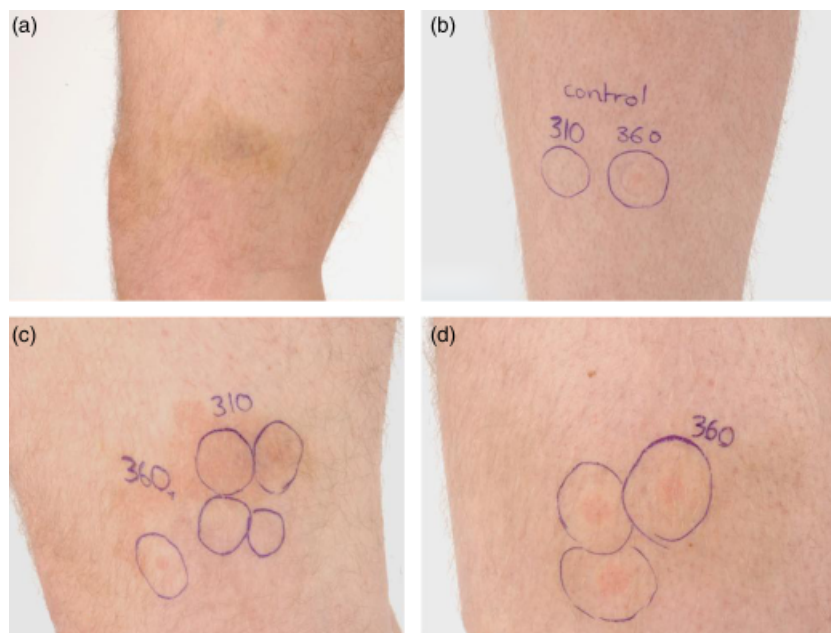


Fig. 1. Pre- and post phototesting; control and bruised sites (310 & 360nm).

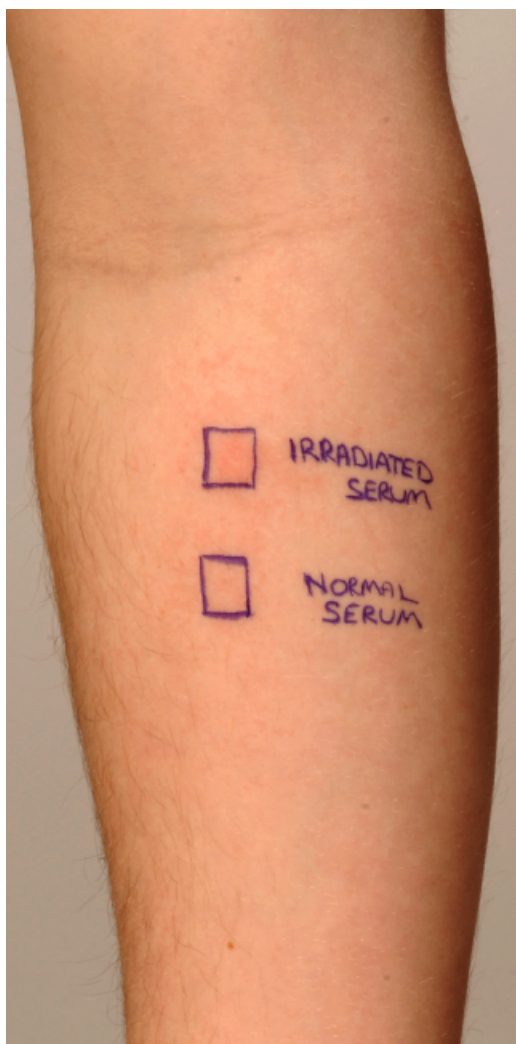


Fig. 2. Reaction to autologous injections of normal and pre-irradiated serum (310nm).

cases with associated systemic features such as wheezing, syncope, and even anaphylaxis (4). Bruising and solar urticaria has once been reported in the literature. Norris and Hawk (5) described a 53-year-old gentleman who described an exaggerated susceptibility to whealing in bruised skin areas. They were unable to elicit any urticarial response on the skin of the upper back with narrow wavebands of 300–600 nm light but demonstrated immediate whealing when repeated in sites of mild bruising at 400–500 nm. They postulated that an increased local concentration of this serum would render bruised skin especially sensitive.

Our patient again showed no abnormal responses with monochromator phototesting on normal looking skin. The positive reaction following the irradiated autologous serum injection confirms a serum factor is responsible in his case. We hypothesize that the vascular leak, resulting from local bruising, leads to an increased local higher concentration of a circulating chromophore. On exposure to UV radiation, the photoallergen is recognized by specific IgE molecules leading to the degranulation of mast cells. The search for the responsible photoallergen in this complex condition continues.

References

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