Hydroa vacciniforme

Case Report

This 8-year-old boy had a 2-year history of recurrent vesicular eruptions localized on the nose and ears. The lesions first appeared in spring, 1-2 days after solar exposure. During the second year, recurrences were observed in the summer months and then again in February of the next year. The lesions also occurred on the forearms; furthermore, healing produced varioliform scars. In March 1993, a particularly severe eruption was observed after sun exposure during a ski camp in the mountains. It affected the face (fig. 1), ears and arms without any sign of systemic illness. The family history was non contributory, in particular there were no known photosensitivity diseases.

On admission, the cheeks, nose and ears were covered by many black crusts, partially confluent, partially isolated, rounded, 0.5-1 cm in diameter. Their borders were erythematous. Bacteriological examination showed the presence of nonhemolytical streptococci, cultures for herpes virus were negative. Urinary and fecal porphyrins were normal, and there were no antinuclear antibodies detected in the serum. The minimal erythema dose was within the normal range for UVB (0.2 J/cm², Sylvania F75 UV6 tubes) and moderately lowered for UVA (5 J/cm², Sylvania FR90 T12). Repeated daily exposure of buttock skin (3x10 J/cm² and 4x5 J/cm²) did not reproduce the clinical lesions.

Based on history, clinical picture and varioliform scarring (fig. 2), the dermatosis was diagnosed as HV. The boy was treated with chloroquine, 100 mg/day, associated with application of a topical broad-spectrum sunscreen. With this treatment, he remained free of lesions. Follow-up extended over 6 months including the summer period.
Fig. 1. The cheeks, nose and chin were covered by many black crusts, partially confluent. Their borders were erythematous.

Discussion

The most recent review on HV comprised 10 cases observed within 27 years in England [1]. This review confirmed that the clinical picture of HV is fairly uniform. HV is characterized by crops of vesicles appearing in about 2 days after sun exposure, leading to varioliform scar formation.

Our case had a history and a clinical picture typical of HV. Phototesting of reported cases gave diverse results [2-6]. In our patient there was a moderate decrease in the minimal erythema dose for UVA, a result which corresponded to some of the previously reported cases [1, 6]. However, we could not induce the formation of typical HV lesions with repeated exposures, as were reported by some authors [5-9]. The pathogenesis of HV is still unknown. Some authors suspected a deficit in vitamin B6, consequently altering tryptophan metabolism; however, vitamin B6 substitution did not improve the symptoms [2, 10]. Treatment is often unsatisfactory, measures occasionally reported to be of some benefit were antimalarials, ß-carotenes and eventually phototherapy [1-3, 11]. Our patient is reported because of his unusually good response to chloroquine 100 mg/day and application of a topical wide-spectrum sunscreen.

Fig. 2. The lesions healed with varioliform scarring.

References


