Papulovesicular eruption located on the face and extremities in a child

A 14-month-old male child presented to our outpatient clinic with pruritic vesicles that had started a month ago, first on the face and then on the hands and feet. With detailed history obtained from his mother we learned that he had had similar complaints in the newborn period, during the last summer, and again during the spring of the present year. She said that his lesions had healed on each occasion, leaving white spots. The family history was negative for photosensitivity.

Dermatological examination revealed a papulovesicular eruption on the face and extremities



Figure 1: Papulovesicular eruption on the face

Figure 3: Superficial orthokeratosis, spongiosis, irregular acanthosis, epidermal degeneration, and minimal perivascular chronic inflammatory cell infiltration in the upper dermis (H and E, \times 100)

How to cite this article: Çoban M, Kocabas E, Temiz P, Ertan P, Ermertcan AT. Papulovesicular eruption located on the face and extremities in a child. Indian J Dermatol Venereol Leprol 2011;77:627-8.

Received: November, 2010. Accepted: February, 2011. Source of Support: Nil. Conflict of Interest: None declared.

and hypopigmented scars on the dorsal aspect of his hands [Figures 1 and 2]. The skin type of the patient was type II. The complete blood cell count and the liver and renal function tests were found to be in the normal ranges. The direct microscopic examination for *Sarcoptes scabiei* was negative. Histopathological examination of skin biopsy showed superficial orthokeratosis, spongiosis, irregular acanthosis, epidermal degeneration, and minimal perivascular chronic inflammatory cell infiltration in the upper dermis [Figure 3].

WHAT IS YOUR DIAGNOSIS?



Figure 2: Papulovesicular eruption and hypopigmented scars on the hands and feet

The diagnosis is hydroa vacciniforme

Hydroa vacciniforme (HV) is a rare, acquired photodermatosis of unknown etiology, which predominantly affects children. It is characterized by the occurrence of vesicles on light-exposed areas, especially the malar areas, bridge of the nose, lips, ears, and the dorsa of the hands and forearms, a few hours after sun exposure. The vesicles crust and heal over a period of 1–6 weeks, leaving varioliform scars.^[1] In our patient, with the detailed history obtained from his mother, we learned that he had had similar complaints last summer and, again, this spring. On dermatological examination, his lesions were localized on light-exposed areas and there were hypopigmented scars. The diagnosis of hydroa vacciniforme can be made on the basis of these clinical and histopathological findings.

HV was first described by Bazin in 1862.^[2] The condition is seen equally in both sexes. Presentation is usually during the first decade, but there is a late-onset variety.^[3] There are no diagnostic laboratory tests for the disease, and the diagnosis is made principally on clinical grounds, supported by the very characteristic histology and the frequently abnormal monochromatic irradiation tests, which demonstrate photosensitivity.^[4]

The early lesion is an epidermal multilocular vesicle with reticular degeneration. A dense, perivascular, lymphohistiocytic infiltrate with vessel hemorrhage and thrombosis may occur in the dermis. Late lesions show epidermal and dermal necrosis with a surrounding, chronic, inflammatory infiltrate. Direct immunofluorescence study results are usually normal, except for rare reports (two patients) of C3 deposits at the dermoepidermal junction and in the small dermal vessels.^[5] Complications of HV are rare. Eye involvement manifests as conjunctivitis, sometimes associated with severe chemosis. Corneal ulcerations have been described in a few cases and occur on the exposed areas of the cornea, with the upper photoprotected surface spared.^[1] Some reports have recently demonstrated that HV is associated with Epstein-Barr virus (EBV) infection and lymphoma, but some of these cases showed atypical features, and these cases may not represent the usual form of HV.^[6]

History, examination, and laboratory investigations are usually sufficient to differentiate these conditions and accurately diagnose HV.^[1] The differential diagnosis of HV includes several blistering disorders that are light induced, for example, erythropoietic protoporphyria (EPP), vesicular polymorphous light eruption (PMLE), bullous lupus erythematosus (BLE), solar urticaria, Hartnup disease, actinic prurigo, hydroa aestivale, porphyria cutanea tarda (PCT), and some forms of xeroderma pigmentosum (XP).^[2,6] The eruptions in EPP are typically an intensely edematous, urticarial reaction, and only its more severe purpuric and vesicular forms cause scarring.^[6] Negative porphyrins in the patients exclude EPP and other porphyrias that can mimic HV, such as congenital erythropoietic porphyria and childhood PCT. Actinic prurigo shows scarring and severe cases may show involvement of non-sunexposed sites such as the buttocks.^[2] Hydroa aestivale is considered by some investigators to be a childhood type of PMLE, while other researchers have postulated that it is a nonscarring form of HV. Unlike HV, PMLE is usually nonscarring and has a later age of onset than HV.^[5] Hartnup disease may clinically mimic HV but is excluded by the absence of aminoaciduria. Bullous LE is unlikely with a normal autoantibody profile. Milder forms of XP such as complementation group F (XPf) are characterized by repeated episodes of erythema with scaling, freckling, mild xerosis, and seborrheic keratosis-like lesions. Blistering is an uncommon feature and scarring does not seem to occur.^[2]

In most patients the condition disappears by the time they reach adolescence, although the scars are permanent. Photoprotection with appropriate clothing and a broad-spectrum sunscreen form the standard approach. If there is no response to photoprotection, spring-time prophylactic therapy using broad-band UVB, narrow-band UVB, or PUVA can be beneficial. Although some anecdotal evidence exists to support the use of beta-carotene/canthaxanthin, azathioprine, and antimalarial therapy, their real value is uncertain. Prophylactic fish oil may also be of help, although poor tolerance limits its use.^[3]

We prescribed a broad-spectrum sunscreen and a topical corticosteroid cream and adviced appropriate clothing for our patient.

Mutlu Çoban, Engin Kocabaş, Peyker Temiz¹, Pelin Ertan², Aylin Türel Ermertcan

Celal Bayar University, Faculty of Medicine, Departments of Dermatology, ¹Pathology and ²Pediatrics, Manisa, Turkey

Address for correspondence: Dr. Aylin Türel Ermertcan, Celal Bayar Üniversitesi Tıp Fakültesi, Dermatoloji Anabilim Dalı, 45010 Manisa, Türkiye. E-mail: draylinturel@hotmail.com

Access this article online	
Quick Response Code:	Website:
	www.ijdvl.com
	DOI: 10.4103/0378-6323.84088

REFERENCES

1. Yesudian PD, Sharpe GR. Hydroa vacciniforme with oral

mucosal involvement. Pediatr Dermatol 2004;21:555-7.

- 2. Gupta G, Mohamed M, Kemmett D. Familial hydroa vacciniforme. Br J Dermatol 1999;140:124-6.
- Hawk JL, Young AR, Ferguson J. Cutaneous Photobiology. In: Burns T, Breathnach S, Cox N, Griffiths C, editors. Rook's Textbook of Dermatology, 7th ed. Oxford: Blackwell Publishing; 2004. p. 1-24.
- 4. Lysell J, Edström WD, Linde A, Carlsson G, Svennilson MJ, Westermark A, *et al.* Antiviral therapy in children with hydroa vacciniforme. Acta Derm Venereol 2009;89:393-7.
- Sebastian QL, Rosario RD. Hydroa vacciniforme: Differential diagnoses and workup. eMedicine Specialties, Dermatology, Photo-Related Diseases. Updated: Jul 24, 2008.
- 6. Park HY, Park JH, Lee KT, Lee DY, Lee JH, Lee ES, *et al.* A case of hydroa vacciniforme. Ann Dermatol 2010;22:312-5.