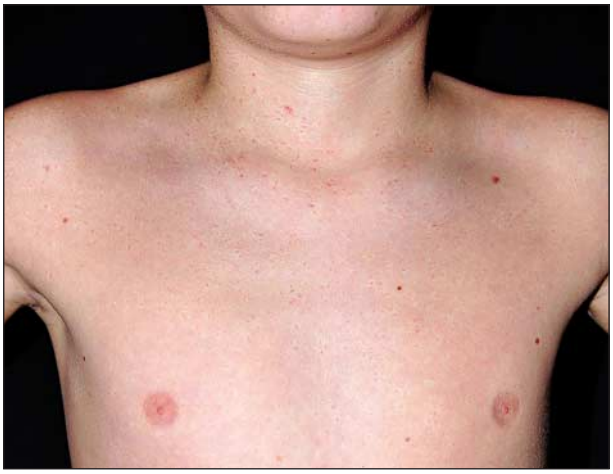

Figure 1. Punctate telangiectasias surrounded by anemic halos on the upper trunk.

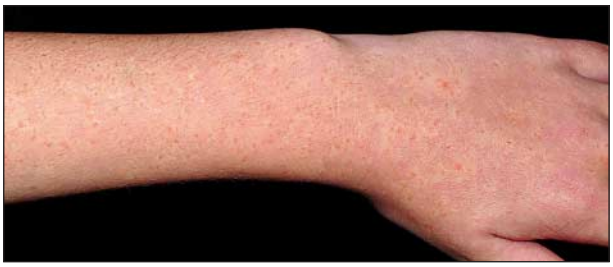

Figure 2. Punctate telangiectasias surrounded by anemic halos on the left arm.

examination showed any signs of bleeding diathesis, systemic involvement, or any other abnormality. The mother had similar lesions in the anterior thorax, which she had not previously noticed. She did not know if there was a family history of lesions. The routine laboratory parameters, including platelet count and the coagulation tests, were all within normal limits. Histopathologic study showed dilation of the subpapillary venous plexus with decreased vascularity of papillae and very little perivascular lymphocytic inflammatory infiltrate.

Discussion. Hereditary benign telangiectasia is an unusual entity.[2] It affects predominantly women (it may worsen during pregnancy[1]) and children between birth and adolescence.[3] However, there are some reports of congenital HBT,[1,4] as is the case with our patients. Interestingly, our younger patient is a boy. There are very few reports of anemic halo punctata telangiectasia.[5] It belongs to the HBT category and is a very well defined entity. Clinically, we can observe asymptomatic, punctate telangiectasias surrounded by a pale halo, localized mainly in the cephalic extremity, anterior and posterior upper trunk, and upper limbs. It probably has an autosomal dominant inheritance pattern.[4,5]

Our juvenile patient had the punctate telangiectasias surrounded by an anemic halo from birth. In contrast, his mother did not realize that she had similar lesions until she attended our service.

The mechanism by which the anemic halo develops remains unclear.[5] Histopathologic findings are unspecific: marked dilation of the subpapillary venous plexus with decreased vascularity of papillae.[1]

There are numerous differential clinical and histopathologic diagnoses of HBT: hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease), angioma serpiginosum, essential generalized telangiectasia, telangiectasia macularis eruptiva perstans, unilateral nevoid telangiectasia, nevus araneus, Fabry disease (especially the telangiectatic type observed in women), and cutaneous collagenous vasculopathy.[4,5]

Owing to its lack of systemic involvement, HBT has a good prognosis.[2] Laser therapy, sclerotherapy, or the combination of both are usually the treatment of choice. However, intense pulsed light is an effective and safe approach.[3]

We believe that no therapeutic intervention is needed for our patients.

We present 2 patients with punctate telangiectasia with an anemic halo to illustrate an HBT variant seldom mentioned in the medical literature.

Nini Yesenia Arévalo, MD
Juliana Martínez del Sel, MD
Lucila Donatti, MD
Myriam Dahbar, MD
Hugo Cabrera, MD
Miguel Allevato, MD

Author Affiliation: Dermatology Division, Hospital de Clínicas "José de San Martín," Buenos Aires, Argentina. **Correspondence:** Dr Allevato, Dermatology Division, Hospital de Clínicas "José de San Martín," Córdoba 2351, Fourth Floor, Buenos Aires PC 1120, Argentina (divisiondermatologia@gmail.com).
Conflict of Interest Disclosures: None reported.
Additional Contributions: We are indebted to José Casas, MD, for histopathologic analysis and Mariano Ortiz, MD.

1. Bosch R, González A, Herrera E. Telangiectasia hereditaria benigna. *Actas Dermo-Sif*. 1991;10:695-698.
2. Javidi Z, Maleki M, Mashayekhi V, Nahidi Y, Omidvar Borna A. Hereditary benign telangiectasia: first case in Iran. *Int J Dermatol*. 2006;45(7):828-830.
3. Purcell E, Condon C. Intense pulsed light therapy in the management of hereditary benign telangiectasia. *Br J Plast Surg*. 2004;57(5):453-455.
4. Nakajima I, Okuyama R, Terui T, Tagami H, Aiba S. The first report of nonhereditary benign telangiectasia. *J Eur Acad Dermatol Venereol*. 2006;20(10):1329-1331.
5. Ujiie H, Kodama K, Akiyama M, Shimizu H. Hereditary benign telangiectasia: two families with punctate telangiectasias surrounded by anemic halos. *Arch Dermatol*. 2010;146(1):98-99.

Use of Sunlight to Treat Dyshidrotic Eczema

Dyshidrotic eczema is a common complaint. It is very often chronic with recurrences. Although no direct cause of the condition has been identified, immunologic reactions are suggested as a symptom-generating mechanism.[1]

Report of a Case. A 49-year-old white man, employed as a teacher, presented with recurrent dyshidrotic eczema of the palms. During the course of the eczema, 5 dermatologists in 2 institutions treated the condition, and all confirmed the diagnosis. On 3 occasions, mycological examination was performed, and on 1 occasion, the patient underwent epicutaneous testing with European standard series allergens, all with negative results. The

Figure 1. Patient palm before exposures to sunlight.

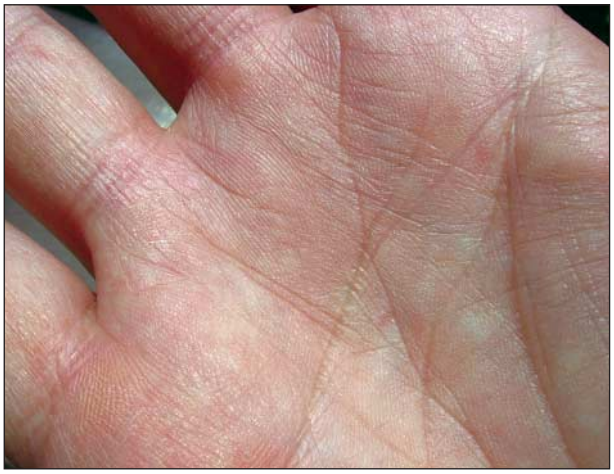

Figure 2. Patient palm after exposures to sunlight and no other treatment.

eczema was treated over a 3-year period, predominantly with topical corticosteroids. Two recurrences were treated with systemic corticosteroids, and 2 recurrences with psoralen plus UV-A irradiation (PUVA). Each course of therapy was successful resulting in the complete disappearance of dermal changes, but recurrences occurred after every course of therapy. The patient experienced several recurrences of dyshidrotic eczema per year. Exposures to sunlight[2] were introduced. Over the next 6 years, the same regimen was followed.

If dermal changes emerged between March and September, the patient exposed his palms to sunlight for 12 minutes every day (other parts of the body were in the shade) when there was direct sunshine between 11:00 and 15:00 hours at a northern latitude of 45° (Turin, Italy; Minneapolis, Minnesota) and at an altitude below 300 m. If the symptoms became distracting, topical corticosteroids were added to his regimen. General measures advised for such patients, minimal hand washing, avoidance of soaps, and avoidance of direct contact with household cleaning products, fresh fruit, and other items, were continued without modification.[3] Exposures to sunlight were discontinued when the changes cleared. In the second year topical corticosteroids were no longer needed. Seven recurrences were successfully treated exclusively with exposures to sunlight (altogether 205 exposures). It usually took 10 to 15 exposures before an improvement was noticed.

Figure 1 shows skin changes just before the start of exposures to sunlight. **Figure 2** shows completely cleared skin after sunlight exposures. Exposure to sunlight was the only intervention.

Discussion. Therapeutic interventions that have an immunosuppressive effect (PUVA or UV-A1 irradiation) are efficient. The immunosuppressive effects of UV irradiation have 2 peaks. One is at 310 nm and belongs to UV-B, and the other is at 370 nm and belongs to UV-A1.[4,5] While the immunosuppressive effect of UV-B irradiation is proportional to the dose of radiation used, the immunosuppressive effect of UV-A1 therapy has a bell-shaped dependency curve, so beyond the peak, the immunosuppressive effect is reduced as the dose of radiation increases.[4] It is estimated that the immunosuppressive effect of UV-A1 is 3-fold that of UV-B during moderate daily exposure to sunlight.[5] The duration of the exposure to sunlight was determined to be below 1 minimal erythemal dose for the maximum intensity of solar radiation at any latitude up to 300 m in altitude and in the range of the maximum immunosuppressive effect of solar UV-A1.[2,4]

The palms are never intentionally exposed to sunlight and unintentional exposure is negligible, so additional exposure is excluded.

Milorad Letić, MD, PhD

Author Affiliation: Department of Biophysics, School of Medicine, University of Belgrade, Belgrade, Republic of Serbia.
Correspondence: Dr Letić, Institut za biofiziku, Medicinski fakultet, Univerzitet u Beogradu, Visegradska 26/II, 11000 Beograd, Republic of Serbia (milet@rcub.bg.ac.rs).
Conflict of Interest Disclosures: None reported.
Funding/Support: This study was supported in part by the Ministry of Education and Science of the Republic of Serbia, project No. 175 036.

1. Storrs FJ. Acute and recurrent vesicular hand dermatitis not pompholyx or dyshidrosis. *Arch Dermatol*. 2007;143(12):1578-1580.
2. Letić M. Exposure to sunlight as adjuvant therapy for dyshidrotic eczema. *Med Hypotheses*. 2009;73(2):203-204.
3. Wollina U. Pompholyx: a review of clinical features, differential diagnosis, and management. *Am J Clin Dermatol*. 2010;11(5):305-314.
4. Halliday GM, Byrne SN, Damian DL. Ultraviolet A radiation: its role in immunosuppression and carcinogenesis. *Semin Cutan Med Surg*. 2011;30(4):214-221.
5. Damian DL, Matthews YJ, Phan TA, Halliday GM. An action spectrum for ultraviolet radiation-induced immunosuppression in humans. *Br J Dermatol*. 2011;164(3):657-659.

Flexural Agminated Eruptive Nevi in Langerhans Cell Histiocytosis

Langerhans cell histiocytosis (LCH) is a multisystemic disease of childhood characterized by abnormal clonal proliferation of Langerhans cells, with skin lesions often involving flexural areas. We report 2 cases of eruptive nevi seen in skin folds of children with LCH.