- Prick test with pollens (grasses, olive, plane tree, Arizona cypress, and weeds such as mugwort, amaranth, *Chenopodium*, *Salsola*, pellitory, and plantain.
- Prick-prick testing with propolis (resinous mix present in beehives), honey from Leon provided by the patient, another honey from Burgos, and a commercial honey with the trade name ''Luna de Miel'' (Honeymoon).
- Prick test with profilin, pollen from birch and oak (as a representative of PR-10), lipid transfer protein from peach, mustard, and sesame (as representatives of storage proteins).
- Laboratory analysis (ImmunoCap, Thermo Fisher): total IgE, specific IgE to honey, bee venom (*Apis* species), as well as to the different pollens available (including some compounds), namely, *Ambrosia elatior*, mugwort, *Parietaria judaica*, *Salsola kali*, chestnut, rBet v 1 (birch PR-10), rBet v 2 (profilin), rPhl p 1 and 2 (timothy grass), and rOle e 1 (olive).

Relevant sensitization was demonstrated to honey with a positive prick test result for the honey from Leon (15 mm, positive results were also recorded for the other 2 honeys) and increased specific IgE to honey (5.93 kU/L) and bee venom (1.77 kU/L). The results of the remaining tests, including the skin tests and specific IgE to pollens, were all negative, except for plantain, although this had no clinical relevance owing to its low probability of involvement.

Primary sensitization in honey-allergic patients may be via honey itself, the components of bee venom (and other bee components), and airborne pollens in the honey,[2–5] which is the most frequent cause and is associated mainly with sensitization to pollen from the compound family (most commonly mugwort) and may vary according to location and season.[1,2,5] IgE to bee venom is detected in 30% of honey-allergic patients, and IgE to hymenoptera is detected in 20% of the general population, although the association between allergy to honey and allergy to bee venom is debatable.[3] In the present case, the patient was sensitized to bee venom with no clinical relevance, since she had only experienced a local reaction to bee stings.

Therefore, the patient was diagnosed with contact urticaria induced by honey that was probably associated with an unidentified protein of the honey itself. Given the patient's refusal to undergo oral challenge, she was recommended to avoid consuming honey.

In such cases, it is important to perform a complete allergy work-up in order to identify the cause and eventually provide the patient with specific, tailored recommendations on avoidance.

Conflicts of interest

The authors declare that they have no conflicts of interest.

References

1. Blanco Guerra C, Ramos García T, Díaz Perales A. Síndromes de reactividad cruzada en la alergia a los alimentos. In: Dávila González IJ, Jáuregui Presa I, Olaguibel Rivera JM, Zubeldia Ortuño JM, editors. Tratado de Alergología, Vol III, 2ª ed España: Ergon; 2015. p. 1049–65.
2. Aguiar R, Cabral Duarte F, Mendes A, Bartolomé B, Pereira Barbosa M. Anaphylaxis caused by honey: a case report. Asia Pac Allergy. 2017;7:48–50.
3. Cifuentes L. Allergy to honeybee... not only stings. Curr Opin Allergy Clin Immunol. 2015;15:364–8.
4. Vezir E, Kaya A, Toyran M, Azkur D, Dibek Mısırlıoğlu E, Kocabaş CN. Anaphylaxis/angioedema caused by honey ingestion. Allergy Asthma Proc. 2014;35:71–4.
5. Helbling A, Peter C, Berchtold E, Bogdanov S, Müller U. Allergy to honey: relation to pollen and honey bee allergy. Allergy. 1992;47:41–9.

L. González-Bravo[a,*], A. González Moreno[a], C. Fuente Sarró[b], E. Gómez de la Fuente[b]

[a] *Unidad de Alergia, Hospital Universitario Fundación Alcorcón, Madrid, Spain*
[b] *Unidad de Dermatología, Hospital Universitario Fundación Alcorcón, Madrid, Spain*

[*] Corresponding author.
E-mail address: lgonzalezb@fhalcorcon.es (L. González-Bravo).

16 October 2019 15 January 2020

https://doi.org/10.1016/j.adengl.2020.01.014

Atypical Palmoplantar Pityriasis Rosea[☆]

Pitiriasis rosada atípica palmoplantar

To the Editor:

A 26-year-old man with no relevant history was seen at the emergency department for asymptomatic palmoplantar lesions that had appeared 3 weeks earlier and had not responded to topical prednicarbate treatment (twice daily for 20 days). The patient reported no fever or systemic symptoms. He had no history of oral or genital ulcers in the preceding weeks or months, and reported no risky sexual relations. Physical examination revealed erythematous oval plaques, some of which showed fine collarette scaling, located on the palms (Fig. 1), soles, and lateral aspects of the feet (Fig. 2). Histology showed superficial lymphocytic perivascular dermatitis with minimal epidermal exocytosis associated with mild spongiosis (Fig. 3). Immunohistochemistry for *Treponema pallidum* was negative. Serological screening using chemiluminescence immunoassay to detect total antibodies against *T pallidum* was initially negative.

[☆] Please cite this article as: Martín-Alcalde J, Elosua-González M, Pinedo-Moraleda FJ, López-Estebaranz JL. Pitiriasis rosada atípica palmoplantar. Actas Dermosifiliogr. 2021;112:372–374.

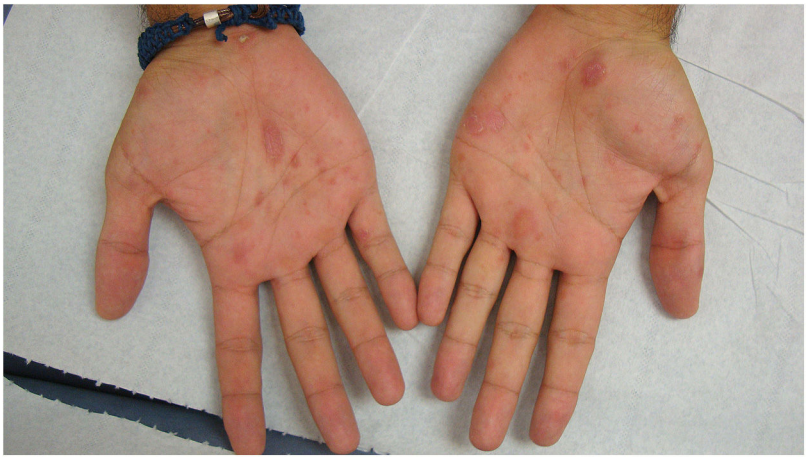

Figure 1 Palmar lesions.

Screening was repeated 1 month later, together with visually interpreted treponemal and non-treponemal tests (*T pallidum* hemagglutination assay [TPHA] and rapid plasma reagin [RPR] tests), all of which were negative. The lesions resolved without treatment after 4 weeks, and the patient remained free of lesions during follow-up, which ended when he again tested negative in a *T pallidum* screening test 3 months after lesion resolution. Based on the clinical course and the clinical, histological, and laboratory data, a diagnosis of atypical palmoplantar pityriasis rosea (PR) was established.

PR is a common entity that mainly affects adolescents and young adults: 75% of cases are diagnosed between the ages of 10 and 35.[1] Clinically, it presents as a papulosquamous eruption with a self-limiting course, distributed mainly on the trunk and the proximal aspect of the extremities, following the Langer lines. These lesions are usually preceded by a larger scaly lesion called a herald patch, and some patients may report prior flu-like symptoms.[1] The literature includes infrequent reports of atypical forms,

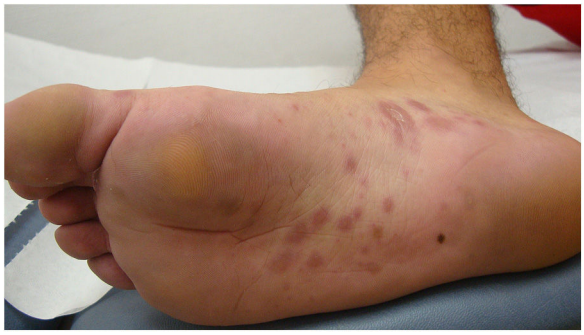

Figure 2 Plantar lesions.

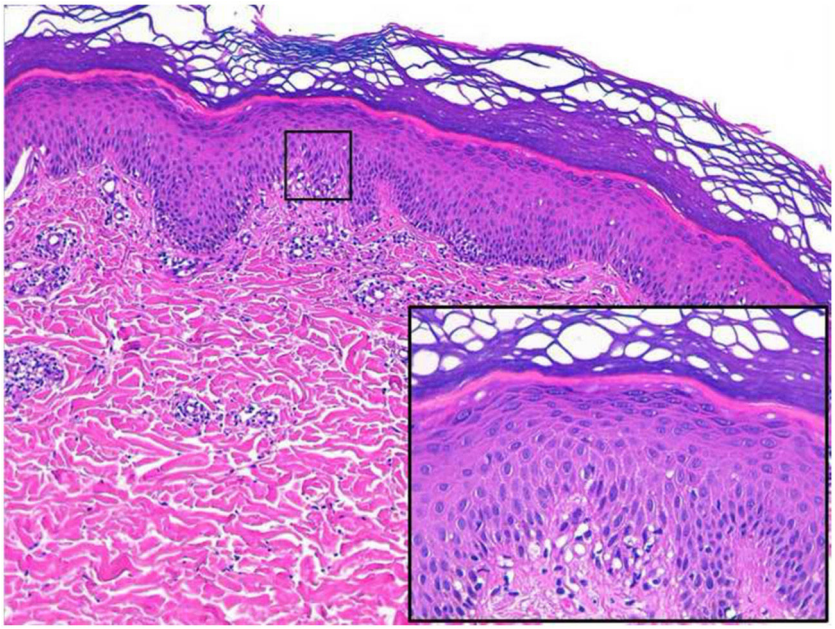

Figure 3 Histological section in which spongiosis (black box) is evident.

characterized by lesions that are morphologically distinct or appear in other locations. These atypical forms include vesicular, purpuric, inverse, unilateral, and palmoplantar PR. Palmoplantar involvement in PR is very rare, and very few cases are described in the literature. In some such cases the palms and soles are affected in the context of a more typical eruption on the trunk.[2,3] Others consist of palmoplantar involvement in the form of vesicular lesions,[4] or of more typical, exclusively palmoplantar lesions.[5] We consider our case to correspond to the latter group, diagnosis of which can be difficult to establish. In all cases of PR with palmoplantar involvement the main differential diagnosis is secondary syphilis. For this reason, serological and histological approaches were used to rule out secondary syphilis in our patient and help establish diagnosis. Histology of PR is nonspecific. In our patient biopsy revealed findings that could be considered compatible with an eczematous process. However, given the clinical appearance of the lesions, the absence of pruritus, and the resolution without treatment, this entity was excluded from the differential diagnosis.

Treatment of PR is controversial. Some data support treatment with erythromycin.[6] However, given the natural course of the disease alternative options include symptomatic treatment of pruritus with topical corticosteroids or oral antihistamines and therapeutic abstention, which was selected in the present case.

We present a case compatible with palmoplantar PR, a rare variant of PR of which very few cases are described in the literature. Despite their infrequent nature, atypical variants of PR can simulate other conditions, and therefore knowledge of these entities is of the utmost importance.

References

1. Chuang TY, Ilstrup DM, Perry HO, Kurland LT. Pityriasis rosea in Rochester, Minnesota, 1969 to 1978. J Am Acad Dermatol. 1982;7:80–9.
2. Deng Y, Li H, Chen X. Palmoplantar pityriasis rosea: two case reports. J Eur Acad Dermatol Venereol. 2007;21:406–7.
3. Bukhari I. Pityriasis rosea with palmoplantar plaque lesions. Dermatol Online J. 2005;11:27.
4. Singh V, Sharma M, Narang T, Madan M. Vesicular palmoplantar pityriasis rosea. Skinmed. 2012;10:116–8.
5. Zawar V. Acral pityriasis rosea in an infant with palmoplantar lesions: a novel manifestation. Indian Dermatol Online J. 2010;1:21–3.
6. Sharma PK, Yadav TP, Gautam RK, Taneja N, Satyanarayana L. Erythromycin in pityriasis rosea: a double-blind, placebo-controlled clinical trial. J Am Acad Dermatol. 2000;42:241–4.

J. Martín-Alcalde[a,*], M. Elosua-González[a], F.J. Pinedo-Moraleda[b], J.L. López-Estebaranz[a]

[a] *Servicio de Dermatología, Hospital Universitario Fundación Alcorcón, Alcorcón, Madrid, Spain*
[b] *Servicio de Dermatología y de Anatomía Patológica, Hospital Universitario Fundación Alcorcón, Alcorcón, Madrid, Spain*

* Corresponding author.
E-mail address: jmartina@fhalcorcon.es (J. Martín-Alcalde).

23 August 2019 26 March 2020

https://doi.org/10.1016/j.adengl.2020.03.018

Good Response to Tofacitinib in Refractory Amyopathic Dermatomyositis☆

Dermatomiositis amiopática refractaria con buena respuesta a tofacitinib

To the Editor:

Dermatomyositis (DM) is an idiopathic inflammatory myopathy characterized by muscle inflammation, skin involvement, and systemic manifestations that are often a very broad spectrum in nature.[1] Amyopathic DM, also known as DM sine myositis, affects a subset of DM patients and presents with cutaneous symptoms but without myopathy.[2]

Our patient was a 49-year-old white woman with a history of thalassemia, for which she had been in follow-up for 7 years. She presented with asthenia, itching, and skin lesions located predominantly in photo-exposed areas. Physical examination revealed heliotrope erythema, Gottron papules, Gottron sign, shawl sign, V sign, and Holster sign. A detailed medical history was taken and a systemic review performed, as well as multiple additional tests, including nuclear magnetic resonance imaging of the pelvic girdle, an electromyogram, a muscle biopsy, and determination of muscle enzymes. All results were normal, and the patient was diagnosed with amyopathic DM. Screening for antinuclear, anti-synthetase, anti-TIF-1γ (transcription intermediary factor 1 γ), and anti-MDA-5 (melanoma differentiation-associated protein 5) antibodies and occult tumors was negative throughout the follow-up period.

Owing to poor disease control, the patient had undergone multiple treatments to which she responded poorly, including topical and oral corticosteroids (maximum dose, 1 mg/kg/d), azathioprine, methotrexate, hydroxychloroquine, mepacrine, rituximab, oral tacrolimus, intravenous immunoglobulins, and mycophenolate mofetil. She was being simultaneously treated with oral prednisone, hydroxychloroquine, mepacrine, and mycophenolate mofetil up until 7 months before the consultation. Given the progressive worsening of her condition and the severity of the skin lesions (Fig. 1) and pruritus, only treatment with low-dose prednisone (2.5 mg/d) was maintained, and tofacitinib (TOF) treatment (5 mg/12 h) was started.

Clinical improvement was observed 2 weeks later and, at the time of writing, all skin lesions have improved significantly (Fig. 2) and the pruritus has disappeared.

☆ Please cite this article as: Crespo Cruz A, del Boz J, Romero Gómez C. Dermatomiositis amiopática refractaria con buena respuesta a tofacitinib. Actas Dermosifiliogr. 2021;112:374–376.