nosis should include inflammatory acne, Favre-Racouchot syndrome, milium cysts, inflammatory tinea, and nevus comedonicus.[1]

When comedones are absent, this disease may be called lupus with an acneiform scarring pattern. In this entity, the lesions are characterized by pitting, resulting from the destruction of the hair follicle and the sebaceous glands due to the inflammatory infiltrate.[3,5–7]

The diagnosis is confirmed by means of the histopathology study, which shows a predominant interphase pattern with hydropic degeneration of the basement layer and thickening of the basement membrane, accompanied by keratotic plugs and comedones, as reported in our patient.[1–3] When histology findings are not conclusive, immunofluorescence may contribute to the diagnosis, revealing a deposit of IgM, IgG, and C3 at the dermal-epidermal junction.[1,6]

Several therapeutic options exist, although the treatment of choice is oral hydroxychloroquine (200 mg/12 h).[1,7] In our case, hydroxychloroquine could not be administered due to the patient's ophthalmologic history. Oral and topical retinoids may be used as an alternative and may be supplemented with topical or intralesional corticosteroids.[1] It should be noted that these treatments presented little therapeutic efficacy in our patient and minocycline (100 mg/d for 3 months) and topical tretinoin, together with strict photoprotection, was indicated, with good results; we therefore propose this regimen as another alternative treatment. The outcome of comedonic lupus is uncertain and, while few patients have been described in the literature, a risk of progression to SLE has been observed in half of cases.[1–3] It should be noted that our patient already presented systemic lupus when the comedonic lupus lesion appeared.

Comedonic lupus thus represents a rare presentation of chronic cutaneous lupus erythematous. Early diagnosis and long-term follow-up are very important due to the risk of systemic progression of the disease.

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Conflicts of Interest

The authors declares that they have no conflicts of interest.

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Hives After Handling Honey☆

Urticaria de contacto por miel

To the Editor:

Contact urticaria consists of the appearance of pruritic hives immediately after contact between the skin and an eliciting agent. It resolves after a few minutes or hours, once exposure has ceased. This condition may involve an immunoglobulin (Ig) E–mediated hypersensitivity mechanism (immunologic origin), although it may also be due to direct stimulation of subcutaneous inflammatory cells or blood vessels by histamine-releasing or vasoactive substances in foods (nonimmunologic origin).[1]

Allergy to honey is rare, with an estimated incidence in the general population of less than 0.001%.[2] The most common symptom is oral pruritus, whereas contact urticaria is an extremely rare symptom.[3]

A 25-year-old woman with seasonal pollinosis as the only item of interest in her personal history consulted for onset of hives immediately after handling honey from beehives on her farm in Leon, Spain. The farm had a large number of oak and chestnut trees. The hives appeared only at the contact areas and disappeared spontaneously after 10 minutes. The patient reported that consuming a small amount of honey did not produce symptoms. She also reported having experienced bee stings with an exclusively local reaction and no concomitant systemic symptoms.

Given the suspicion of allergy to honey, a series of additional tests were performed, as follows:

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- 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.

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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.

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