dyskeratotic dermatoses. Genetic analysis remains to be performed in our patient, but the coexistence of PAD and BFP in the same patient supports this hypothesis.

We have described a patient with the simultaneous presence of lesions clinically and histologically typical of BFP and PAD; the study of mutations in both lesions remains to be performed. To date, we have found no case reports in the literature describing the coexistence of BFP and PAD in a single patient.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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1578-2190/

Bullous Phytophotodermatitis Caused by an Esoteric Remedy☆

Fitofotodermatitis ampollosa producida por un remedio esotérico

To the Editor:

The patient was a 33-year-old woman with a past history of hypothyroidism, anorexia nervosa, and anxiety-depression with episodes of self-harm. She was on treatment with levothyroxine, fluoxetine, clonazepam, and clomethiazole, and there had been no recent changes in her medication. She attended the emergency department for a 3-day history of large, painful blisters on the dorsum of both hands and fingers (Fig. 1). The blisters contained a serous fluid and the surrounding skin was erythematous and pruritic. The patient denied having applied any substance to the area or contact with plants, and at that moment she was off work because of her psychiatric illness and dedicated her time to walking and reading outside, even though it was winter. The lesions were reminiscent of large burns and, suspecting that they may have been self-inflicted, it was decided to admit the patient to observe the clinical course. During her admission, she was prescribed topical therapy with fusidic acid and betamethasone, and oral therapy with prednisone and amoxicillin–clavulanic acid, leading to an improvement in the lesions within a few days. On resolution of the acute bullous condition, we observed very clearly defined, diffuse pigmentation of a residual appearance on the skin distal to the wrists (Fig. 2), but that did not affect the area covered by a ring; this suggested a possible diagnosis of phototoxicity. Additional tests performed, including extensive blood tests with autoimmune studies and 24-hour urinary porphyrin levels, determination of the minimal erythematous dose for UV-A and UV-B, and patch and photopatch testing with the standard series of the Spanish Contact Dermatitis and Skin Allergy Research Group and photoallergens of the Spanish Photobiology Group, were rigorously normal. Histology of the lesions showed a subepidermal blister with epidermal necrosis. Occasional apoptotic keratinocytes were observed in areas adjacent to the blister and a dermal infiltrate of lymphocytes, histiocytes, and eosinophils, with a number of extravasated red blood cells were observed in areas adjacent to the blister; these findings were compatible with a diagnosis of bullous phototoxic dermatitis. On further questioning, the patient finally remembered having applied a product prepared by a faith healer to combat an evil-eye curse 24 to 36 hours prior to onset of the lesions. This remedy consisted of an infusion of a plant called *rue*, which the patient had to apply all over her body except on her head. The site of the lesions coincided with the only area that had not been covered by her winter clothing after applying the substance.

Plants of the genus *Ruta* are small bushes originating from southern Europe that are cultured as ornamental plants in gardens and also for their medicinal properties and as a condiment. Among their many effects (abortifacient, antiparasitic, insect repellent, analgesic,...), they are widely known for their phototoxic capacity due to the furanocoumarins (5-methoxypsoralen and 8-methoxypsoralen) and alkaloids that they contains.[1] Numerous cases have been published of phytophotodermatitis due to the topical application of distinct species of *Ruta* for various therapeutic uses, such as pediculicide[2] or analgesic[3] lotions, or direct use of the plant as an insect repellent.[4] In most cases, the clinical diagnosis is simple because of the site of the lesions in

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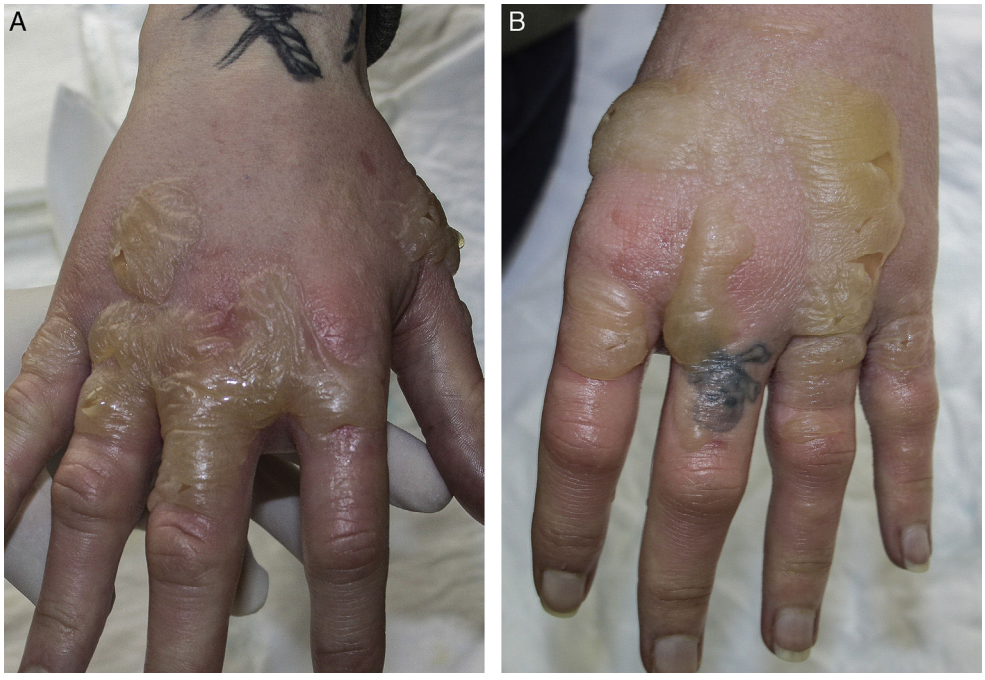

Figure 1 A, Erythema and edema on the dorsum of the right hand, with large tense blisters with a transparent content. B, Similar lesions on the dorsum of the left hand.

sun-exposed areas, the tendency to form vesicles or blisters, and the residual pigmentation, together with a history of contact with the plant or the application of substances that the plant contains.

Blister formation in the phytophotodermatoses can be intense, simulating extensive burns.[5] In children, contact with phototoxic plants can be accidental, when playing in gardens, and is often not remembered.[6] The appearance of lesions similar to

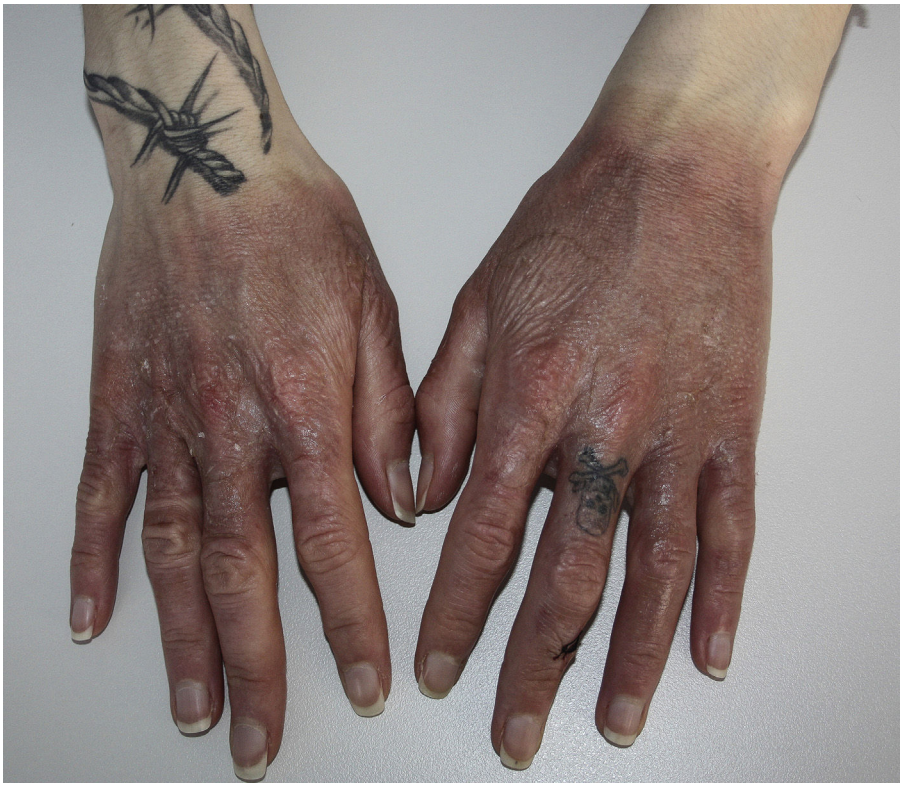

Figure 2 Diffuse pigmentation on the dorsum of the hands after resolution of the lesions. The clear limit at the distal borders of the wrists and the absence of involvement of the area under the ring on the fourth finger of the right hand are very striking.

burns with no history of contact with plants can confound the diagnosis.

Apart from its therapeutic applications, *Ruta* is used widely in some countries of the Iberian peninsula and Latin America to protect against ''evil spirits''.[7,8] The recommendation to apply products containing this plant all over the body, typically followed by exposure to the sun, perhaps even on the beach, gives rise to widespread lesions that only spare the areas covered by the bathing suit. Severe episodes of extensive phytophotodermatitis require a multidisciplinary approach with supportive measures in burns units.

We have presented a case of bullous phytophotodermatitis due to the use of *Ruta* in an unusual esoteric remedy against an evil-eye curse. A diagnosis of phytophotodermatitis must be suspected in patients with bullous lesions only affecting sun-exposed skin. On resolution, the lesions tend to produce well-defined residual pigmentation. It is also important to consider this diagnosis in children, in whom contact with the plant may be accidental and pass unnoticed.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Coma Blisters After an Overdose of Central Nervous System Depressants☆

Ampollas del coma tras sobredosis de fármacos depresores del sistema nervioso central

To the Editor:

The term *coma blisters* refers to a condition that occurs in patients who lose consciousness. It was first described in 1812 by Larrey[1] in soldiers with carbon monoxide poisoning. Since then, it has been mainly associated with overdose of drugs and nervous system depressants, such as barbiturates, tricyclic antidepressants, opiates and alcohol; neurological disorders, such as meningoencephalitis, cerebrovascular disease, and cranioencephalic trauma; and metabolic disorders, such as hyperkalemia, hypoglycemia, and diabetic ketoacidosis.[2–4] Coma blisters, however, have also been described in patients without an altered state of conscience, in particular in cases of long immobilization or Wegener granulomatosis.[5–7]

We present the case of a 24-year-old woman with a history of a personality disorder and occasional consumption of cocaine and amphetamines who was found unconscious in her home. She had taken multiple pills from her regular medication supply (topiramate, duloxetine, quetiapine, and clorazepate).

On arrival at the emergency department, she had a low level of consciousness (score 6 on the Glasgow Coma Scale), pale skin, and reactive mydriatic pupils. Partial improvement (Glasgow Coma Scale 10) was observed following physical stimulation, and the patient was treated with oxygen, fluid therapy, gastric lavage, and activated charcoal.

There was no evidence of acute intracranial lesions on the computed tomography scan. The laboratory workup showed a serum creatine kinase level of 5590 U/L and normal kidney function. The urine drug screening test showed high levels of benzodiazepines.

During her first 24 hours in hospital, the patient developed asymptomatic skin lesions located mainly on bony prominences. The physical examination showed tense clear fluid-filled blisters on well-delimited erythematous plaques (Fig. 1A,B). The lesions had an artifactual morphology and were characteristically located on pressure points (metacarpophalangeal joints on the right hand, right hip, and left knee).

The histopathologic examination showed a subepidermal blister with foci of reepithelialization (Fig. 2) and focal epithelial necrosis of eccrine coils, with periglandular infiltration of neutrophils (Fig. 3A). Additional findings included dermal, perivascular, and periadnexal infiltrates, which were predominantly neutrophilic, together with foci of fibrinoid necrosis in the walls of the small dermal capillaries and neutrophilic infiltration of the walls (Fig. 3B).

Administration of topical antibiotics led to resolution of the lesions within 3 weeks, and there were no signs of scarring or recurrence.

The clinical presentation and histopathologic findings were consistent with a diagnosis of coma blisters.

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