

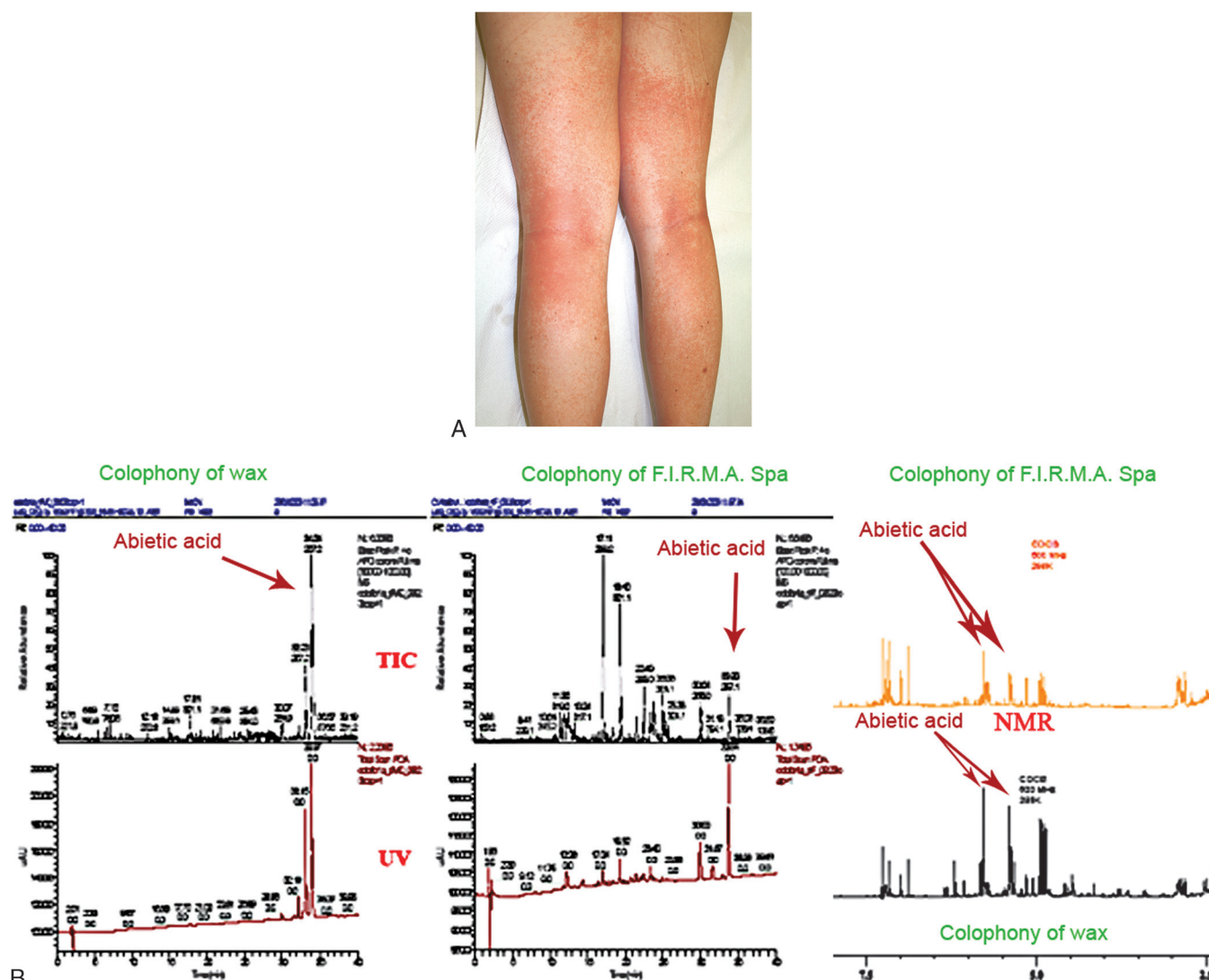


Figure 1 . A, Allergic contact dermatitis of the lower limbs after wax epilation. B, Chemical analyses (HPLC, mass spectrometry, ultraviolet spectroscopy, NMR) performed on 2 types of colophony (colophony of wax and colophony of F.I.R.M.A. S.p.A.) show an almost similar pattern of signals, but with a different quantitative ratio.

PEARL

Allergy to colophony in cosmetics can be misunderstood with patch testing only the standard series. To avoid false-negative results, use products and their constituents should be tested.

REFERENCES

- Downs AM, Sansom JE. Colophony allergy: a review. *Contact Dermatitis* 1999;41:305–310.
- Sadhra S, Foulds IS, Gray CN. Identification of contact allergens in unmodified rosin using a combination of patch testing and analytical chemistry techniques. *Br J Dermatol* 1996;134:662–668.
- Karlberg AT, Gäfvert E. Isolated colophony allergens as screening substances for contact allergy. *Contact Dermatitis* 1996;35:201–207.
- Goossens A, Armingaud P, Avenel-Audran M, et al. An epidemic of allergic contact dermatitis due to epilating products. *Contact Dermatitis* 2002;47:67–70.
- Quain RD, Militello G, Crawford GH. Allergic contact dermatitis caused by colophony in an epilating product. *Dermatitis* 2007;18:96–98.

Scalp “Sensitivity” Without Visible Lesions

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PRECIS

We present a case of “pruritus” of the scalp without visible lesions that was unresponsive to conventional treatment modalities.

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DISCUSSION

A 63-year-old man presented with a 4-year history of itching restricted to the scalp. Clobetasol 0.05% solution was the only effective medical intervention, although this gave him relief for no more than 1 hour after application. The patient stated that the application of rubbing alcohol to affected areas was the only other effective temporary treatment. Physical examination and histopathology revealed no specific abnormalities. Patch testing to 46 standard allergens was negative. Treatment with topical corticosteroids, oral antihistamines, antibiotics, and neuromodulatory medications failed to alleviate the “pruritus.”

Most patients who complain of itching of the scalp present with visible lesions or dermatoses, from which a clinical or histological diagnosis can usually be made. Common causes of such scalp dermatoses include seborrheic dermatitis, contact dermatitis, psoriasis, dermatomyositis, and pediculosis. By contrast, patients with “pruritus” of the scalp without visible lesions also commonly present for relief of symptoms. A literature search of PubMed and Ovid databases led us to use “sensitivity” instead of just “pruritus” as the most encompassing terminology of symptoms from patients’ perspectives.

The pathophysiology of skin sensitivity without visible lesions is poorly understood with an apparent reduced “tolerance threshold” of skin for itch but without specific immune or allergic reaction present and histopathology limited (if at all) to vasodilation or some subtle inflammatory response.¹ Often noted, however, is increased transepidermal water loss that may allow greater entry of irritants through the skin.¹

In contrast to other body areas, histamine may not be a mediator of itching on the scalp because in vivo analysis revealed decreased vascular and sensory sensitivity to histamine-induced pruritogenesis on the scalp as compared with the forearm, which in turn may be due to the scalp having significantly fewer histamine-sensitive chemonociceptors.² Central processing of the itch stimulus could also be different, requiring a higher central threshold for itching on the scalp versus the area subserved by the trigeminal nerve.² Pain may play a role in masking perceptions of itch as well because opioid administration often results counterintuitively in “pruritus” of the scalp.²

Up to 70% of patients with skin sensitivity have a psychiatric illness,³ and it is well known that psychiatric illnesses and psychosomatic factors can elicit “pruritus” and affect perception of scratching behavior.⁴ Furthermore, the scalp and face are the most common sites of involvement in localized skin sensitivity associated with psychiatric disease.³

Unfortunately, no high-level data exist to guide clinical practice in the treatment of scalp sensitivity. Topical corticosteroids and oral antihistamines often fail to relieve the “pruritus,” perhaps because of the scalp’s different local mediator of itch or different central processing of itch stimulus. More efficacious treatment modalities have been shown to focus on treatment of comorbid psychiatric disorders with use of tricyclic antidepressants, selective serotonin reuptake inhibitors, anxiolytics, and opioid antagonists.^{2–5}

PEARL

Our case highlights the difficult challenge of managing scalp sensitivity without visible lesions. Our literature review leads to a recommendation to include psychiatric specialists for consultation and consider use of antidepressant therapy for such cases.

REFERENCES

1. Misery L, Sibaud V, Ambronati M, et al. Sensitive scalp: does this condition exist? An epidemiological study. *Contact Dermatitis* 2008;58:234–238.
2. Rukwied R, Zeck S, Schmelz M, et al. Sensitivity of human scalp skin to pruritic stimuli investigated by intradermal microdialysis in vivo. *J Am Acad Dermatol* 2002;47:245–250.
3. Ferm I, Sterner M, Wallengren J. Somatic and psychiatric comorbidity in patients with chronic pruritus. *Acta Derm Venereol* 2010;90:395–400.
4. Schneider G, Driesch G, Heuft G, et al. Psychosomatic cofactors and psychiatric comorbidity in patients with chronic itch. *Clin Exp Dermatol* 2006;31:762–767.
5. Hoss D, Segal S. Scalp dysesthesia. *Arch Dermatol* 1998;134:327–330.

Prevention of Airborne Propolis-Induced Allergic Contact Dermatitis With Barrier Cream

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PRECÍS

Airborne contact dermatitis from propolis can occur in nonbeekeepers and may be preventable with use of a barrier cream.

CASE REPORT

A 34-year-old woman was referred for evaluation of an intermittent pruritic rash during spring and summer for 4 years, occurring on her face, neck, arms, knees, and anterior thighs (Figures 1 and 2). Lesions generally appeared 2 days following work in her yard, whether or not sunscreen and insect repellent were applied. Previous biopsy showed spongiosis with superficial and middermal perivascular infiltrate with eosinophils. Her allergist determined allergies to dust and mold but not grasses and pollens. The patient described no relief from topical corticosteroids or oral antihistamines, but gradual spontaneous resolution. Patch tests with North American Extended Series-65 and 15 additional allergens (Chemotechnique Diagnostics AB, Malmö, Sweden) using Finn Chambers (Epitest Ltd Oy, Tuusula, Finland) and Scanpor tape (Norgeplaster Alapharma A/S, Vennesla, Norway)

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