

Isothiazolinones in paint as a cause of airborne contact dermatitis in a patient with psoriasis receiving anti-interleukin-17 therapy

Constanza Martinez-Mera  | Maria A. González | Mercedes Hospital | Lucia Turrión-Merino

Department of Dermatology, Hospital Universitario Puerta de Hierro, Majadahonda, Madrid, Spain

Correspondence

Dr Constanza Martinez-Mera, Dermatology Department, Hospital Universitario Puerta de Hierro, Majadahonda, Manuel de Falla 1, 28222, Majadahonda, Madrid, Spain.

Email: c.martinezmara@gmail.com

KEYWORDS: airborne dermatitis, allergic contact dermatitis, anti-interleukin-17, interleukin-17, isothiazolinones, ixekizumab, paints, treatment

The immunological mechanism of allergic contact dermatitis (ACD) is based on the interaction between the innate and the adaptive responses. Some studies have described the presence of elevated levels of interleukin (IL)-17 in ACD patients.^{1–3}

CASE REPORT

A 66-year-old man with no known allergies was referred to the dermatology department. He had been diagnosed with plaque psoriasis without arthritis 8 months previously. His Psoriasis Area and Severity Index (PASI) was 12, and his Dermatology Life Quality Index was 12. He started taking ixekizumab at the usual dose, and his PASI decreased to 0 after 8 weeks. At week 20, he presented with pruritic, erythematous and oedematous lesions on the face but not on the body. He associated this with sleeping in his freshly painted house the previous day. Suspecting airborne ACD, we advised him to sleep

elsewhere for at least 6 weeks, and prescribed deflazacort 30 mg daily (tapering dose) and topical methylprednisolone aceponate 0.1% for 2 weeks (Figure 1).

After 2 months, patch testing was performed with the TRUE Test series (Marti Tor, Barcelona, Spain) and with the four paints originally used in the house “as is” in a semi-open test. Readings on day (D) 2 and D4 showed positive reactions to methylisothiazolinone (MI), methylchloroisothiazolinone (MCI)/MI, paint 2 and paint 4. MCI/MI was a component of both paints. The clinical features and the results of the patch test led us to diagnose airborne ACD caused by MCI/MI in paints.

DISCUSSION

Ixekizumab is a humanized IgG4 monoclonal antibody that binds to IL-17A and blocks the interaction with its receptor, IL-17RA. IL-17 plays a stimulatory role in the sensitization and effector stages,⁴ and has been

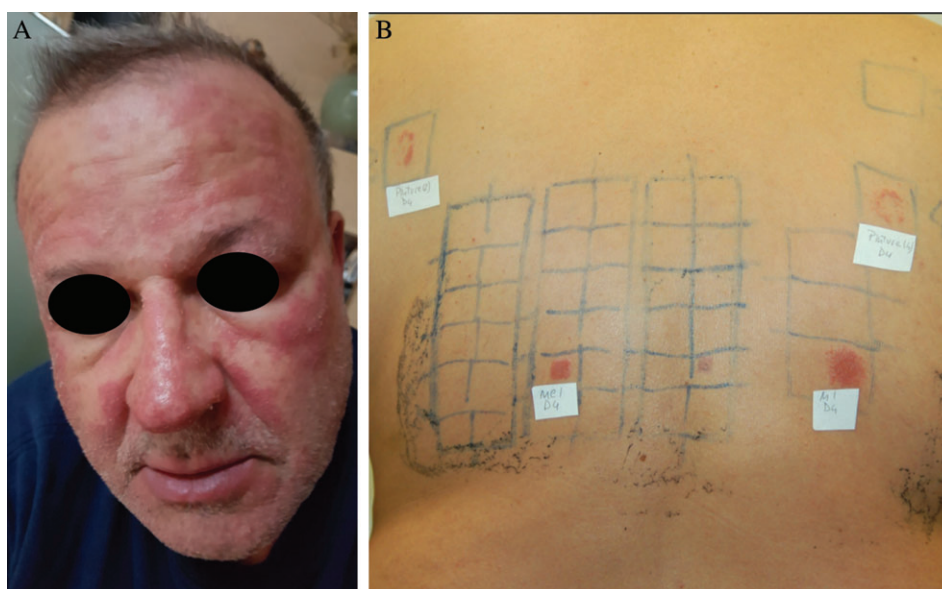


FIGURE 1 (A), Pruritic, erythematous and oedematous facial lesions within 24 hours of onset. (B), Day 4: patch test results were positive for methylchloroisothiazolinone (MCI)/methylisothiazolinone (MI), MI, paint 2 and paint 4

reported to be elevated in patients with ACD caused by nickel and fragrances.^{1–3} In addition, T helper 17 memory cell counts were higher in the peripheral blood of nickel-allergic individuals than in control patients.³ Another report showed that sensitization with contact allergens induces a long-lasting local memory response and a temporary global immunological memory response to the allergen that is mediated by IL-17A and interferon- γ produced by memory T cells.⁵ A recent study suggested that the blockade of this route could be used to treat ACD, although the authors recruited only three patients, and observed minor or no improvement in eczema following treatment with secukinumab.⁶ In the case reported, we were unable to confirm this hypothesis, as the patient was receiving an anti-IL-17 agent, to which his psoriasis responded very favourably, but still developed airborne ACD. After 8 months of follow-up, the patient has followed the recommendations and has not developed new lesions suggestive of ACD. His PASI remains at 0. In conclusion, the findings of Todberg et al⁶ and those of the present case are preliminary, although they probably point to a more complex mechanism in the immunoregulation of ACD.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to report.

ORCID

Constanza Martinez-Mera  <https://orcid.org/0000-0002-2824-4205>

REFERENCES

1. Martin SF. Immunological mechanisms in allergic contact dermatitis. *Curr Opin Allergy Clin Immunol*. 2015;15:124-130.
2. Peiser M. Role of Th17 cells in skin inflammation of allergic contact dermatitis. *Clin Dev Immunol*. 2013;2013:1-10.
3. Larsen JM, Bonefeld CM, Poulsen SS, Geisler C, Skov L. IL-23 and TH17-mediated inflammation in human allergic contact dermatitis. *J Allergy Clin Immunol*. 2009;123:486-492.e1.
4. Silvestre MC, Sato MN, Reis VMDS. Innate immunity and effector and regulatory mechanisms involved in allergic contact dermatitis. *An Bras Dermatol*. 2018;93:242-250.
5. Schmidt JD, Ahlström MG, Johansen JD, et al. Rapid allergen-induced interleukin-17 and interferon- γ secretion by skin-resident memory CD8⁺ T cells: local memory response to contact allergens. *Contact Dermatitis*. 2017;76:218-227.
6. Todberg T, Zachariae C, Krustup D, Skov L. The effect of treatment with anti-interleukin-17 in patients with allergic contact dermatitis. *Contact Dermatitis*. 2018;78:431-432.

How to cite this article: Martinez-Mera C, González MA, Hospital M, Turrión-Merino L. Isothiazolinones in paint as a cause of airborne contact dermatitis in a patient with psoriasis receiving anti-interleukin-17 therapy. *Contact Dermatitis*. 2019;80:328–329. <https://doi.org/10.1111/cod.13203>