To the Editor:

Atopic dermatitis is a multifactorial chronic inflammatory disease whose unpredictable clinical course is influenced by a complex genetic basis (atopic diathesis) and multiple triggering factors. In adults, atopic dermatitis is characterized by severe pruritus, xerosis, lichenified plaques of eczema, and excoriation. No curative treatment is yet available. Specific immunotherapy with house dust mite allergens has recently been reported to be effective in adult patients with severe atopic dermatitis who are sensitized to these allergens.1

We describe a 25-year-old man who came to our clinic in January 2006 for a severe flare-up of atopic dermatitis over 40% of his body. He presented an objective SCORing Atopic Dermatitis (SCORAD) score of 39.5 and a total SCORAD score of 55.5 (Figure 1). The laboratory workup showed a total serum immunoglobulin E (IgE) of 2919 kU/L and alanine aminotransferase of 192 U/L. Relevant history included chronic atopic dermatitis from childhood, persistent rhinitis, childhood asthma (currently asymptomatic), recurrent facial herpes, and chronic liver abnormality of unknown origin. The patient usually followed treatment consisting of topical emollients, corticosteroids, and tacrolimus, plus nonsedating antihistamines and oral acyclovir. The acute flare-up was treated by adding sedating antihistamines, oral antibiotics, and oral corticosteroids with clear improvement. Phototherapy with narrow-spectrum UV-B was subsequently started, but was switched due to lack of effectiveness to psoralen-UV-A baths for 2 months, with excellent response. Patch testing with the standard GEIDAC panel from the Spanish Contact Dermatitis and Skin Allergy Research Group (Grupo Español de Investigación en Dermatitis de Contacto y Alergia Cutánea) was negative at 96 hours, and prick tests with airborne allergens showed sensitization to Dermatophagoidespteronyssinus (Der p, 4 × 4 mm), D. farinae (Der f, 5 × 4 mm), and grass pollens (3 × 3 mm). IgE-specific antibodies against Der p and Der f were above 100 kU/L (Class 6). In September 2006, the atopic dermatitis worsened, with an objective SCORAD score of 30, a total SCORAD score of 45, and a total serum IgE of 7133 kU/L. The patient could not undergo phototherapy, however, and refused oral immunosuppressive therapy. Because he was highly sensitized to house dust mites, a decision was made to use subcutaneous specific immunotherapy with polymerized house dust mite allergens from the Diater laboratory, which includes the Der p 1 (45 %), Der f 1 (45 %), Der p 2 (4.5 %), and Der f 2 (4.5 %) allergens. In November 2006 the patient started the initiation phase, which consisted of weekly administration of increasing allergen doses. From the fourth week, he switched to maintenance therapy at the maximum dose (0.5 mL, allergen mass 2.5 µg) once monthly, and this therapy was ongoing at the time of writing without any adverse effects. From the second month of specific immunotherapy, the patient reported an obvious improvement in his symptoms, reducing the use of topical corticosteroids and tacrolimus and oral antihistamines. One year after the start of specific immunotherapy, the patient had an objective SCORAD score of 8, a total SCORAD score of 17, a total serum IgE of 2290 kU/L, and an overall response to treatment considered to be moderate by the patient and noticeable by the physician (Figure 2). Moreover, the patient had no asthma symptoms, although his persistent rhinitis was unchanged.

Atopic dermatitis has multiple triggers, including house dust mites in sensitized patients. Dermatophagoides feed on human skin scales and are found in large quantities in mattresses, pillows, stuffed animals, sofas, rugs, and carpets. House dust mites can trigger eczema flare-ups by inhalation2 and by skin contact, as shown in patch testing.3 Subcutaneous specific immunotherapy with house dust mite allergens was first used in atopic dermatitis many years ago.
Two clinical studies have been published in the past year on this treatment, in which patients with atopic dermatitis and high house dust mite sensitization (IgE-specific antibodies ≥ 3.5 kU/L, class ≥ 3) were successfully treated with very few adverse effects\(^1\)\(^5\) (Table). In our case, we opted for specific immunotherapy with polymerized allergens because of its excellent efficacy, safety, and convenience profile.

Although a new treatment should not be proposed based on a single case, the authors feel that studies should be conducted to compare the therapeutic effect of specific immunotherapy to that of usual treatments for atopic dermatitis,\(^6\)\(^7\) and that specific immunotherapy with house dust mite allergens should be considered in patients with severe atopic dermatitis who are sensitized to these allergens.

### References