5 mg/D), and a short course of oral corticosteroids (prednisone 1 mg/kg/D) We recorded mild clinical improvement of dermatitis at first month of therapy (SCORAD 71.9) After three months, our patient showed spectacular improvement of symptoms score and quality of life (SCORAD 30.2). Now, he continues with monthly immunotherapy with excellent tolerance. He practices judo without affectation of the skin or exacerbations of eczema. Rhinitis and asthma have shown improvement on symptoms scores and functional tests, too.

In conclusion, we present a case of severe atopic dermatitis in a dust-mite sensitisation patient with excellent response to specific immunotherapy. Although immunotherapy is not considered a first-line treatment for atopic eczema (indeed, in severe cases it is considered a contraindication), we thought that it may be a good alternative[1,2] in patients with demonstrated allergy to environmental agents before the introduction of more aggressive therapies such as cyclosporine.

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Rapid oral desensitisation to prophylactic isoniazid

To the Editor,

Isoniazid is an essential drug in the treatment and prevention of tuberculosis. It is metabolised in the liver by an acetylation process to form major and minor metabolites. The use of isoniazid may lead to hypersensitivity reactions such as fever, rash, eosinophilia, haemolytic anaemia, angiitis, and hepatitis; which may occur singly or in combination.[1] The most common skin lesions are morbiliform, maculopapular or urticarial rash.[2] Some rapid desensitisation protocols for isoniazid have been reported.[2–4] The treatment with adalimumab, an anti-TNF monoclonal antibody, is associated with the reactivation of tuberculosis and requires a prophylactic treatment with isoniazid for nine months.[5]

We report the case of a 56-year-old woman with no previous history of atopic disease or other antibiotic hypersensitivity. She has been followed for rheumatoid arthritis for six years and has been put on meloxicam and sulphasalazine treatment for six years and leflunomide therapy for four years. Three months ago, she started to receive adalimumab injections twice a month. Due to this immunomodulatory treatment, she was advised to receive one isoniazid tablet of 300 mg daily, a drug she had never previously taken, for tuberculosis chemoprophylaxis. The patient noticed generalised itching and erythema on her arms and legs almost 12 h after the first dose; however, ignoring the symptoms, she continued the isoniazid therapy. By the time, the skin lesions developed to the typical urticarial lesions and a mild oedema in the eyelids occurred. Thus, the patient ceased taking izoniazid tablets after four days and, on presentation at the next day, physical examination revealed urticarial and papular lesions on both thighs and excoriation scars on the arms. There were no symptoms and signs concerning other body systems. The vital signs and basic laboratory findings were normal, including the liver function tests. The patient was prescribed an oral antihistamine for a week and the lesions disappeared without trace in three days. No biopsy was taken from the lesions. The patient denied having experienced any previous urticarial skin lesions.

The patient was hospitalised six weeks after the reaction for drug tests and a possible intervention for desensitisation. The informed consent of the patient was obtained. The skin tests for isoniazid were negative (prick tests with 0.1 mg/ml and 1 mg/ml, and intradermal tests with 0.03 ml of 0.1 mg/ml and 1 mg/ml of isoniazid). Although the skin tests did not support an IgE-mediated hypersensitivity reaction, desensitisation was considered in the light of clinical manifestation. A rapid oral desensitisation was performed the next day, following the protocol displayed in the Table 1. No reaction was observed during and after the procedure and the patient was discharged from hospital on a regimen of 300 mg isoniazid daily. Previous reports recommended the use of the injectable form of isoniazid for skin tests and the use of the elixir form for the initial small doses (<50 mg) of desensitisation.[2] In our country, the only available forms of isoniazid are 100 mg and 300 mg pills. Therefore, a suspension was obtained by dilut-

Table 1 The protocol for rapid oral desensitisation.

Time (min)	Isoniazid (mg)	Reaction
0	6.25	–
30	12.5	–
60	25	–
120	50	–
240	100	–
360	150	–
480	150	–
Next day	300	–

ing the pill in distilled water and this was used for both the skin tests and in the desensitisation protocol.

Like most of the previous reports in the literature, the skin tests were negative in our case. This suggests a non-IgE mediated immunologic reaction to isoniazid or that the reaction was caused by a metabolite of isoniazid.[2–4] Rapid desensitisation, a method classically used for type I IgE-dependent hypersensitivity, is also applicable for the cases where the presence of drug-specific IgE was not demonstrated.[6] A recent consensus statement on rapid desensitisation underlined that most of the patients who had experienced hypersensitivity reactions within 24 h after the last dose of drug reached tolerance with this procedure.[7] Traditional desensitisation protocols for isoniazid took longer durations; therefore, they could pose a risk for resistance development. The patient has just concluded the fourth month of the prophylactic isoniazid therapy and, thanks to rapid desensitisation, the patient was able to use the drug without further hypersensitivity reaction and with less risk for resistance development. This case showed that diluted oral pills of isoniazid could be used for desensitisation when other forms were unavailable. To our knowledge, this patient is the first reported case of rapid oral desensitisation to isoniazid used for chemoprophylaxis.

Conflict of interest

None of the authors declared any conflict of interest.

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