exists as to whether the use of intravenous administration of heparin is really safe for all these patients.

In conclusion, we report a rare case of delayed-type hypersensitivity reaction to Bemiparin. We have demonstrated cross-reactivity between Bemiparin and Enoxaparin by patch test. Due to the possibility of cross-reactivity between heparins, before recommending an alternative heparin, an allergic study with patch, intradermal and subcutaneous challenge tests must be performed. We think that subcutaneous challenge test is necessary to confirm the tolerance of the alternative drug.

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Case report: specific immunotherapy with dust mite allergens in a child with severe atopic dermatitis

To the Editor,

Atopic dermatitis is a chronic inflammatory skin condition that appears to involve a genetic defect in the proteins supporting the epidermal barrier. The goals of treatment are to reduce symptoms, prevent exacerbations, and to minimise therapeutic risks. This includes general measures, antihistamines, topical or systemic corticosteroids, topical calcineurin inhibitors and management of infections. In severe cases, systemic immunosuppressive agents like cyclosporine may be useful, but are not exempt of important adverse effects. Although there has been some controversy regarding the role of allergy in atopic dermatitis, the bulk of the data indicate that allergy plays a role in selected patients. Dust mites are consistently the most common positive aeroallergen, and also appear to be the most clinically relevant. However, specific immunotherapy is not generally taken into account as a therapeutic tool for atopic dermatitis.

A 10-year-old male patient with a history of persistent rhinitis and mild asthma was referred to our unit with severe atopic dermatitis, presenting intense pruritus, lichenified plaques, scaly and excoriated papules with huge affectation of quality of life (bad sleep, impossibility to practice sports). SCORAD at first visit was 106.6. Laboratory tests showed IgE levels of 12457 UI/ml with dust mite specific levels >100 kU/ml (*Dermatophagoides pteronyssinus*, *Blomia tropicalis*). He was not sensitised to other environmental or food allergens. Unfortunately, the severity of eczema did not allow the performing of skin prick test or atopy patch test. Treatment with antihistamines, topical and systemic corticosteroids showed only partial response.

We started specific subcutaneous immunotherapy (ALK-ABELLO, *Dermatophagoides pteronyssinus* 60%, *Blomia tropicalis* 40%) in addition to sustained treatment with antihistamines (hydroxyzine 50 mg/D and levocetirizine

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	–