tion, due to its inherent risks, and an interval of at least 4 weeks should be respected until cutaneous testing is performed.

When available, drug specific IgE should be determined, since it is of great value for diagnostic purposes in the work up of drug allergy. However, in our centre the only available specific IgEs are for amoxicillin, penicilloyl G, penicilloyl V, ampicillin and cefaclor (Phadia® catalogue), so, no specific IgE to ceftriaxone was determined in this patient, although it would have been of great importance.

Desensitisation for drug allergy is the induction of temporary clinical unresponsiveness to drug antigens. The main purpose of developing rapid desensitisation protocols for the treatment of drug hypersensitivity is to provide essential medications to patients avoiding severe reactions[4]. The administration of small doses of a drug until achieving the full therapeutic dose can be safely performed in allergic patients, allowing the treatment of severe diseases. These procedures should only be performed in the hospital setting and by fully trained medical personnel. There has been increasing interest in developing more rapid desensitisation protocols to antibiotics, mainly β-lactams, regarding its essential role in a wide number of infections, like syphilis in pregnant women.

There are several protocols for intravenous cephalosporin desensitisation, which range from hours to 14 days. A typical protocol for desensitisation to intravenous penicillin or cephalosporins starts at 1/10000 to 1/100 target dose, doubling the dose every 15-20 minutes, until reaching the full therapeutical dose[5].

Because this patient was seriously ill, it was decided to proceed with a drug desensitisation protocol with close observation and monitoring of the patient, starting with a very small initial dose (0.001 mg) of ceftriaxone. The total dose (1 g/day) was achieved after 3 days and effective treatment was completed until total recovery of the patient (30 days).

Although more recent and rapid desensitisation protocols to cephalosporins are described in literature, the authors applied a 3-day protocol, because of the severity of the illness, and the patient could have more risks with a faster protocol.

No work up study was done to confirm the drug allergy diagnosis since the patient missed the scheduled appointment. However, the clinical history and the response to drug withdrawal clearly points to the diagnosis of drug allergy.

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Exanthematic reaction to pseudoephedrine

To the Editor:

Pseudoephedrine is a sympathomimetic drug widely used in over-the-counter anti-catarrhal preparations. Despite its widespread use, cutaneous adverse effects are rare, and generally not life-threatening.

A 30-year-old Caucasian woman presented with a generalised, maculopapular, pruriginous dermatitis with facial oedema, malaise and fever (Fig. 1). During the five days preceding the emergence of the cutaneous lesions, the patient had been taking Actifed® (pseudoephedrine and triprolidine) for rhinorrhea and nasal congestion with no other accompanying symptoms. She denied previous intake of this drug.

Physical examination revealed a generalised, maculopapular dermatitis, sparing the distal extremities and mucous membranes, with facial oedema. Tympanic temperature was 38 °C. No peripheral lymph nodes were palpable and hepatosplenomegaly was absent.

Laboratory tests revealed slight neutrophilia (7.7×10^9 cells/L) without leucocytosis and elevated C-reactive protein levels in blood (21.2 mg/dL). Mycoplasma pneumonia, Epstein Barr, Cytomegalovirus, B and C hepatitis, HIV-1 and HIV-2 serologies were negative. Antistreptolysin titter was negative. Chest radiography showed clear lung fields.

Histopathological examination revealed vacuolar degeneration of the basal layer, oedema and haemorrhage of the papillary dermis and mixed perivascular inflammatory infiltrate.

The patient's condition was successfully managed with oral prednisolone.

Patch tests performed with Actifed® as it is and with pseudoephedrine sulphate (1% pet) were both positive (++). Portuguese standard series, ephedrine (1% pet) and phenylephrine (1% aq) were negative. We were unable to test for triprolidine. Testing pseudoephedrine sulphate (1% pet) elicited no reactions in five healthy controls.

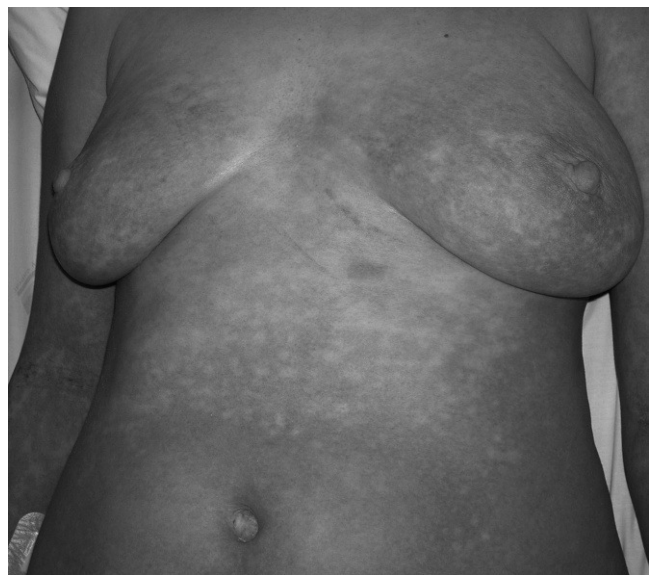

Figure 1. On admission: generalized, maculopapular, pruriginous dermatitis

Pseudoephedrine-induced cutaneous adverse effects are uncommon. Fixed drug eruption accounts for the majority of the cases. However generalised papular and papulovesicular eruption, erythroderma, systemic contact dermatitis, acute generalised exanthematous pustulosis, pseudo-scarlatina and recurrent toxic shock syndrome have also been described[1-6].

Pseudoephedrine belongs to the phenylamine family of compounds and shares with ephedrine a common phenylpropanolamine skeleton. Phenylephrine is also a phenylamine but has a phenylethanolamine skeleton. Allergic contact dermatitis has been reported to phenylephrine eye-drops[7] and ephedrine containing anti-catarrhal preparations[8]. Given that cross-reaction between these two drugs and pseudoephedrine has been described previously[9] it would be prudent to patch test patients whenever pseudoephedrine allergy is suspected. No restrictions were made concerning other sympathomimetic drugs in our patient as no cross-sensitivities were found.

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Snail allergy without house dust mites sensitisation

To the Editor:

Snail consumption is frequent in the Mediterranean area as a delicacy, mostly in Portugal, Spain, Italy and France[1]. It has been recognized as a food allergy with a significant relation with house dust mite (HDM) sensitisation (associated or not with respiratory symptoms). This fact is usually due to cross-sensitisation to the major allergen, tropomyosin, present in HDM, seafood, snail and cockroach[2,3]. Snail allergy can induce several clinical presentations namely asthma, urticaria, angio-oedema, anaphylaxis or even death[1]. There have been very few case-reports of isolated snail allergy, in the absence of HDM sensitisation.

The authors report a 49 year old man, who was admitted to the Immunoallergology Department in 2006 with a first episode of bilateral eyelid oedema, itchy lip and throat, and dysphonia thirty minutes after snail ingestion, although denying either urticaria or dyspnoea. He had no history of respiratory symptoms (namely rhinitis or asthma) suggestive of HDM or any other aeroallergens allergy; he also denies prior food allergy.

The patient was submitted to skin prick tests (SPT) with HDM and other aeroallergens namely cockroach, which were all negative. SPT with snail extract (*Helix aspersa*- Bial Aristegui®) and *prick-to-prick* with cooked and raw snail were positive and negative to all other food allergens, namely shrimp, crab, lobster, clams, cuttlefish, and squid. Specific IgE (Unicap-Phadia®) to HDM and tropomyosin were negative (< 0.35 kUA/L) and positive to snail-*Helix aspersa* (0.89 kUA/L — class II). Total IgE was 66.0 kU/L.

Immunoblotting and inhibition tests of serum sample from the patient were performed in Sweden (MIAB-Phadia®), using Novex® (USA) XCell miniCell, Novex® nitrocellulose blotting membranes and NuPAGE 4-12 % running buffer and rabbit anti-IgE, biotin labelled, in-house, MIAB®. Other equipment used was biotin labelled protein standard, low molecular weight range (phosphorylase b 97 kD, bovine serum albumin 66KD, ovalbumin 45 kD, carbonic anhydrase 31 kD, soybean trypsin Inhibitor 21 kD, Lysozym 14 kD), colorimetric development (BCIP/NBT), BioRad and streptavidin-alkaline phosphates, Zymed®, USA. Two bands, a distinct one at 55 and a faint one at 95 kDa were identified, which were not paralleled by similar bands on the in-house control sample (in-house human pool of sera from snail sensitised patients and our patient samples were immunoblotted against SDS-PAGE separated serial dilutions of extracts from f314 snail, MIAB), *i.e.* they were specific for the patient (Fig. 1).

The absence of HDM sensitisation was confirmed with the determination of native protein of *Dermatophagoides pteronyssinus* (n Der p1) and tropomyosin which was negative. Lack of HDM sensitisation was also supported by inhibition tests, where the IgE binding to snail was inhibited by snail extract but not by the HDM extract.

The peculiarity of this case-report is based on the fact that snail allergy without cross-sensitisation with HDM is very uncommon. Some authors have described some bands of this species of snail (*Helix aspersa)*, but in this patient one (55 kDa) and probably two (95 KDa) new bands were