activity to newly acquired allergens following antihelminthic treatment in migrants from Ethiopia[12]. Together with new environmental factors, which are able to enhance sensitisation, like a polluted atmosphere, this could be the cause for the appearance of new respiratory symptoms in a subgroup of our migrant patients. A previous report proposed a differential atopy evaluation when dealing with the relationship between atopy and parasites[13]. Thus, arthropod-parasite related sensitisation, which includes HDM sensitivity, in our migrant population could have a different clinical outcome compared to pollen or other aeroallergen sensitisation, as is shown by our results.

This reasoning is not contrary to the possible implication of genetic factors. Evidence supports an increased susceptibility to allergy and asthma among populations with tropical ancestry[14]. The differences in asthma prevalences due to ethnicity could thus be due to a common genetic factor that predisposes to both allergy and resistance to infection[15].

Summarizing, the high prevalence of HDM sensitisation of our migrant patients could be due to previous subclinical sensitisation in their tropical and subtropical countries with more favourable conditions for the presence of different mite species, but other factors have to be taken into account, such as genetic factors, the elevated burden of microbial burden or the higher prevalence of geohelminths, which in turn could be responsible for possible cross-reactive sensitisation against HDM.

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Ambroxol-induced systemic contact dermatitis confirmed by positive patch test

To the Editor:

Ambroxol hydrochloride (trans-4-(2-amino-3,5-dibromobenzylamino) cyclohexanol hydrochloride) is a bromexine metabolite[1] (Fig. 1). Both ambroxol and bromexine are well known mucolytics and ambroxol has also been used in clini-

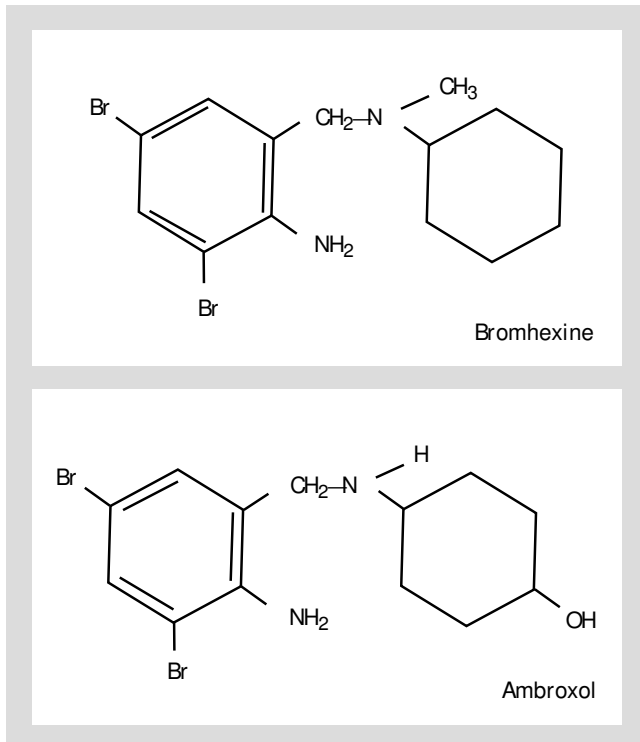

Figure 1. Chemical structures of bromexine and ambroxol.

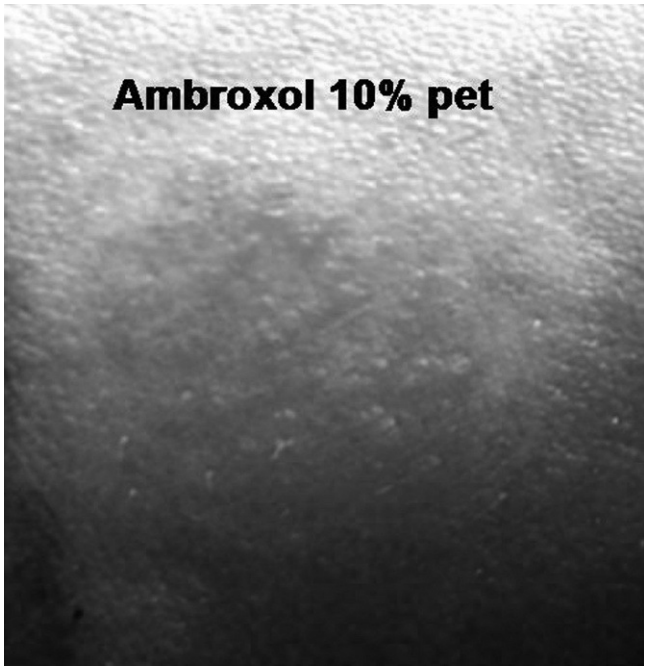

Figure 2. Patch test with ambroxol (10%pet) positive at 96 h (+++).

cal trials for the prevention of chronic bronchitis and respiratory distress syndrome in infant.

A 79-year-old woman presented a maculopapular rash with intense pruritus. She had taken ambroxol, acetaminophen, and codeine for 4 days at doses of 90 mg/day, 1,500 mg/day and 90 mg/day respectively, prescribed for odynophagia. On clinical examination there was a generalised maculopapular exanthematous eruption, with furfuraceous desquamation and intense erythema. The mucosa was spared. She was treated with hydratation, antihistamines, and oral corticosteroids and the skin lesions resolved within a week. After this episode the patient tolerated acetaminophen and acetylsalicylic acid. She had no personal and familiar history of atopic diseases.

Patch tests were performed, according to the guidelines of the International Contact Dermatitis Research Group, with the GEIDC standard series, ambroxol (10%pet) and codeine (10%pet).

Positive reaction was found to ambroxol at 48 hours (++) which increased at 96 (+++) (Fig. 2), and negative to codeine and the standard series. Patch tests were negative in 10 controls. The oral challenge was not carried out for ethical reasons.

Delayed reactions to systemic drugs are not rare and patch test is a well-known method for diagnostic confirmation purposed in patients with generalised type IV hypersensitivity reactions.[1,2] We report a systemic contact dermatitis due to ambroxol, presented as a maculopapular rash and confirmed by patch testing.

Systemic cutaneous reactions by immediate hypersensitivity have been described during the treatment with ambroxol such as urticaria. Delayed reactions to ambroxol such as non-pigmented fixed erythema[3] and contact sensitivity to ambroxol have also been reported when administered by aerosol,[4] but as far as we know, this is the first case of systemic generalised dermatitis caused when taken orally.

Exanthema induced by bromexine has been reported[5] and despite the chemical structures being very similar, the absence of cross-reactivity between them has been demonstrated in one case.[4] More studies should be reported to as certain it definitely. We did not perform an oral challenge with bromexine because of the possibility of inducing a severe reaction, and because it was not an essential drug for the patient.

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Severe allergy to poultry meat without sensitisation to egg proteins with concomitant Leguminosae allergy. Case report

To the Editor:

The number of known animal food allergens is limited. Although chicken meat is quite a common part of European diet and hen's egg is one of he most frequent allergens in children, severe poultry meat allergy without sensitisation to egg proteins is extremely rare.[1-3] Allergy to turkey and duck meat is even more rarely reported and the implicated allergens are poorly characterised.[2-4] In the bird-egg syndrome, sensitisation to chicken feather allergen occurs by the respiratory route and afterwards allergy symptoms appear due to bird meat consumption. The implicated allergen is thought to be alpha-livetin.[2] We report a clinical case of severe chicken and turkey meat allergy without sensitisation to egg proteins. There was also coexisting Leguminosae allergy.

The particular interest of this case is the need for liver transplantation and use of immunosuppressive therapy in the patient with familial amyloid neuropathy.