

old and is doing well. His serum total IgE level remains relatively low (93 IU/mL). Because his *STAT3* mutation is a known mutational hot spot for autosomal dominant HIES, he will be closely monitored for dental, skeletal, and pulmonary complications.

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LATEX: A HIDDEN OCCUPATIONAL ALLERGEN

We present a case of occupational asthma in a 24-year-old man who worked in a butcher's shop. During the past year he developed rhinoconjunctivitis and asthma symptoms related to his work, especially when he went into the back room of the butcher's shop (an adjacent space where a coworker prepared sausages and cut veal bones). The results of physical examination and radiologic and basal spirometric studies were normal. A methacholine inhalation test revealed bronchial hyperresponsiveness (provocation dose that caused a decrease in forced expiratory volume of 20% [FEV₁] of 144 cumulative breath units).

The results of skin prick tests to common aeroallergens, milk, and veal proteins were all negative. The results of prick-prick tests with veal, pork, sausage, a mixture of preservatives and spices used in the manufacture of sausages, cochineal red, carmine red, and powdered veal bone provided by the patient were all negative. The result of a

skin prick test with latex was positive. A handling test with latex gloves was then performed with a negative result.

Blood analysis revealed eosinophilia (11%; eosinophil count of 580/mm³). The total serum IgE level was 323 kU/L. The specific IgE level (ImmunoCAP System; Phadia, Uppsala, Switzerland) for latex was 17 kU/L (reference range, <0.35 kU/L).

The result of a specific bronchial challenge test with an aqueous extract of natural latex (500 µg/mL; ALK-Abelló, Madrid, Spain) was positive with an immediate response (10 minutes after administration of the last dose, 5 inhalations of 50 µg/mL of latex extract, a 28% decrease in FEV₁ was observed).

Air concentrations of latex were analyzed by a volumetric air measuring device (Air Sentinel; ICx Technologies, Arlington, Virginia) at the butcher's shop itself (0.26 ng/m³ of air), the back room of the shop (0.80 ng/m³ of air), and the bedroom of the patient's home (0.09 ng/m³ of air).

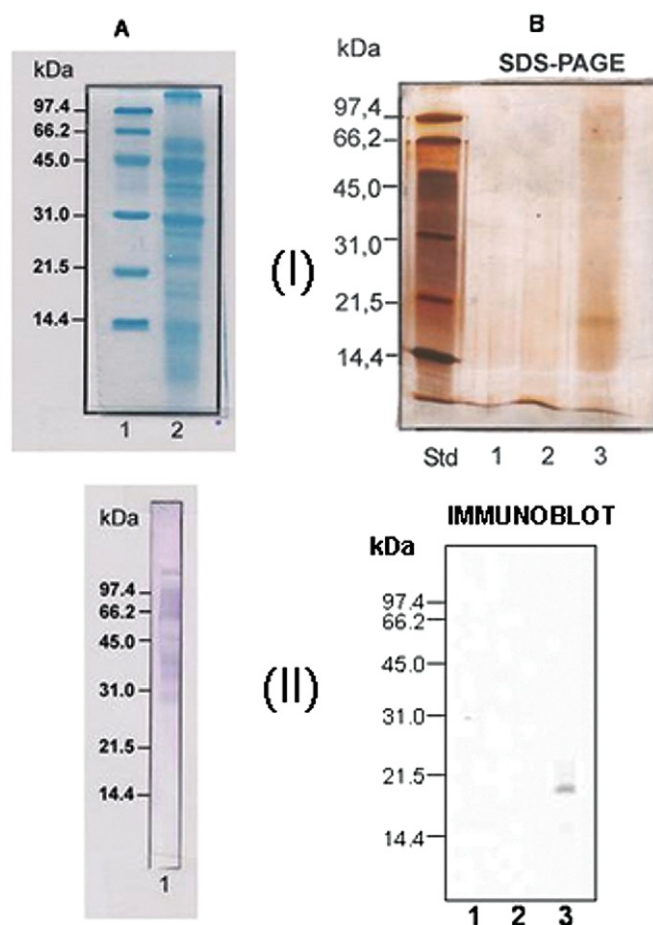


Figure 1. I. A, Sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) analysis. A, Latex extract, 100 µg (Coomassie stain). Lane 1: standard low molecular weight; lane 2: latex extract. B, Extract from filters (silver staining). Lane Std: standard low weight; lane 1: filter butcher's shop; lane 2: filter patient's bedroom; lane 3: filter back room. II. Immunoblot experiments (chemiluminescence). A, Solid phase. Latex extract, 100 µg. B, Solid phase. Extracts from filters. Lane 1: filter butcher's shop; lane 2: filter patient's bedroom; lane 3: filter back room.

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The sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) method visualized several bands between 6 and 100 kDa in a whole latex control extract (Leti Laboratories, Madrid, Spain), but only an 18- to 20-kDa band was visualized in the back room filter extract. No bands were detected in the other filter extracts (Fig 1). Several bands of different allergens were detected in the latex control extract by immunoblot experiments with the patient's serum, indicating sensitization to latex. The same individual band from the back room filter visualized in the SDS-PAGE showed IgE binding capacity. This allergen, with a molecular weight of 18 to 20 kDa, was detected with the patient's serum (Fig 1). IgE inhibition studies demonstrated that the filter from the back room was capable of inhibiting the whole latex extract by 17%, indicating the existence of common allergens. No inhibition was detected with the other filter extracts.

We report a case of occupational asthma in a butcher. The tests performed confirmed that latex was the etiologic agent despite the fact that the patient had no direct contact with latex gloves. Probably the gloves used by the coworker were the source of the latex.

Other reported causes of occupational asthma in butchers were excluded in the present case.^{1–5} Currently, latex is considered to be a potent allergen and 1 of the most frequent etiologic agents in occupational asthma, although the exact prevalence is unknown. In our country butchers are not a group at high risk of latex allergies because they do not usually use gloves.

Recently, there have been reports of latex-induced asthma^{6–8} in individuals who have not used gloves. These reports and the present case confirm that latex is an occupational allergen that may be unnoticed. In such cases, it may be necessary to confirm the presence of significant levels of ambient latex and thus determine the source and intensity of exposure. The threshold for ambient latex necessary to induce sensitization has not been established. Baur et al⁹ found that ambient levels of 0.6 ng/m³ of air in hospitals were associated with an increase in sensitizations and symptoms in sensitized individuals. In our case, there were only clinically significant ambient levels (0.8 ng/m³) in the back room (where a coworker used latex gloves). The levels found in the butcher's shop and the patient's home were not significant. These findings have a direct correlation with the symptoms experienced by the patient.

Commonly, patients with latex allergy also present with contact urticaria. In this case, our patient did not usually use gloves; the route of exposure and sensitization was exclusively airborne. This could explain why the patient had no contact urticaria with latex (negative glove use test result).

In vitro experiments conducted in filters confirmed the existence of latex allergens in suspension. An allergen of 18 to 20 kDa with IgE binding capacity, probably Hev b 5 or Hev b 6 (both major latex allergens), captured in the back room filter and capable of inhibiting the whole latex extract was probably the allergen responsible for asthma in this patient.

In conclusion, we believe that latex should be ruled out in any patient with occupational asthma and especially in those workers with unexplainable allergic reactions related to the workplace even though they report no direct contact with this agent. Prevalence of latex-induced asthma may be greater than expected.

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MAINTENANCE OF REMISSION WITH LOW-DOSE OMALIZUMAB IN LONG-LASTING, REFRACTORY CHRONIC URTICARIA

Omalizumab, an anti-IgE monoclonal antibody, is increasingly being used in diseases other than allergic asthma.^{1–3} We report on the successful long-term management of refractory chronic urticaria in 2 patients using low-dose omalizumab.

The first patient was a 53-year-old woman with a 13-year history of chronic urticaria. She was atopic, with a total IgE serum level of 557 kU/L (reference range, <260 kU/L) and a high titer *Parietaria judaica* specific IgE (>100 kUA/L). Specific IgE to grass pollen was also detected (3.41 kUA/L). Rhinitis, conjunctivitis, and mild asthma were also present but easily managed with inhaled medications and antihistamines. Chronic urticaria, on the other hand, was not responsive to standard medications (antihistamines, leukotriene receptor antagonists, cyclosporine). On physical examination, hives were scattered throughout her body (Fig 1A), including the palms and soles. A search for concomitant causes of chronic urticaria, including infectious agents and parasites, did not yield any clues. An autologous serum skin test (ASST), to screen for chronic autoimmune urticaria, could not be performed because of daily occurrence of hives. To obviate the need for an ASST, donor basophils were challenged with patient serum, with negative results compared with controls (basophil histamine release test). In addition, antinuclear antibodies, antielectable nuclear antigens, cryoglobulins, and antithyroid antibodies were not detected, and complement levels were normal. Thus, she was prescribed omalizumab, 300 mg subcutaneously every other week, based on her total serum IgE levels and body weight. Seventy-two hours after the first anti-IgE administra-

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