Latex is recognized as a cause of occupational asthma (OA) in various working environments. Latex allergy in the textile industry has been recently reported [1]. We herein describe the first case of a seamstress who developed OA to latex.

A 36-year-old woman, a nonsmoker, started work as a seamstress in 1981. She worked with cotton and gabardine. From 1991, she sewed elasticized ribbons, using a different sewing machine, for a few days at a time at irregular intervals, but almost weekly. In 1993, she developed nasal congestion, rhinorrhea and sneezing, and runny and itchy eyes, as well as itchy skin, followed a few weeks later by chest tightness, wheezing, and cough. Symptoms occurred progressively when she worked with the elasticized ribbons, and a few days of exposure also caused nocturnal awakenings. These symptoms improved during weekends and when she was reassigned to other jobs in the same plant. When we first evaluated her in December 1997, after a week of not exposure, FEV₁ had returned to normal (FEV₁/FVC = 2.57/3.19 l, pred 2.67/3.05 l) (2). Methacholine PC₂₀ was positive at 1 mg/ml (48% fall in FEV₁, methacholine PC₂₀ at the end of the day was 0.7 mg/ml). The next day, the subject was exposed to nebulized latex extract (Stallergènes, Paris, France) in increasing doses, starting with a dilution of 1/16 for 60 s up to the undiluted extract for 30 s (the undiluted extract caused a 4.4 mm wheal on SPT and contained 100 IR/ml of latex allergen). This exposure induced a dual asthmatic reaction with an immediate fall in FEV₁ of 20.5% at 20 min after exposure with complete recovery at 50 min, followed by a 16.6% fall at 5.5 h with complete recovery after inhale salbutamol. The next morning, when her FEV₁ was 22% lower than the previous baseline, methacholine PC₂₀ was significantly reduced (0.07 mg/ml). Respiratory symptoms were exacerbated for several days with nocturnal awakenings in the 3 days following SIC; she was given fluticasone 2000 μg/day. By 10 days after exposure, FEV₁ had returned to normal (FEV₁/FVC = 2.86/3.32), and methacholine PC₂₀ was 0.25 mg/ml.

OA to latex is reported increasingly in health-care workers (5) and in other industrial environments [6, 7]. We describe the case of latex allergy in a seamstress who developed rhinoconjunctivitis and asthma after sewing elasticized ribbons. Recently, latex allergy was demonstrated in 2/10 workers in an elasticized ribbon-manufacturing plant, one of them experiencing work-related asthma confirmed by SIC [1]. These workers were sensitized by handling powdered natural rubber, like health-care workers who wore powdered natural rubber gloves, where the latex allergen is adsorbed to the air-suspended powder. Our subject was exposed to rubber in a form that is not usually sensitizing by inhalation, however, sewing the elasticized ribbons induced a fine white dust of cloth and latex that could be inhaled, explaining the route of sensitization.

This case illustrates that one should be mindful of the possibility of latex allergy in different occupational settings.

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References

Specific immunotherapy for occupational latex allergy

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Key words: hypersensitivity; immunotherapy; latex; occupational disease.

Latex allergy in health workers is an increasingly worrisome type of occupational disease. Its prevalence ranges from 12% to 18%, with different symptoms and clinical manifestations, and the disorder represents a serious social and occupational problem for individuals with severe symptoms [1, 2]. An association between latex allergy and hypersensitivity to certain fruits and nuts is also frequently seen [latex-fruit syndrome] [3].

We present a 31-year-old white female patient who had been employed as a radiology technician since the age of 24 years. She reported palpebral edema and burning and itching of the nose and eyes of about 2 years' duration. The symptoms appeared while she was at work, when she spent time in areas where latex gloves had been used. Her symptoms became progressively more severe, with the appearance of intense, sudden-onset urticaria approximately 5 min after exposure in areas of skin that had been in contact with latex gloves. At the same time, she developed severe nasal congestion and obstruction, with prolonged bouts of sneezing. In March 1997, she developed two acute anaphylaxis crises after eating chestnuts and figs, and she developed two acute anaphylaxis crises after eating chestnuts and figs, and she developed two acute anaphylaxis crises after eating chestnuts and figs. On day 65, we initially reduced the once-daily dose to 0.35 cc for 2 more days and increased it again to 0.40 cc (0.4 µg protein) on days 70 and 72. The 0.4-mL dose, on day 72, was the maximum tolerated dose, and was thus used as the maintenance dose. This dose was reached after 20 injections with a cumulative dose of 3.99 µg protein.

Once the maximum tolerated dose was reached [0.40 µg protein], it was given weekly for 5 weeks. The rest of the maintenance dose was given at 2-week intervals. At the time of writing, the patient had been receiving treatment for 5 months, of which time 10 weeks corresponds to the maintenance period. During this period, she received 8 s.c. injections of extract, equivalent to a cumulative dose of 3.2 µg protein. The total cumulative dose of protein received since the start of SIT was 7.19 µg in a total of 28 injections.

We emphasize this patient's excellent adaptation to treatment, and full compliance with the schedule of allergen extract administration and tests. Immunotherapy with this latex extract produced no alterations in the main lymphocyte subpopulations, serum levels of immunoglobulins, and other laboratory values.

Skin tests for latex and food allergens were repeated several times during desensitization, before SIT was started and before the dose of allergen was increased. With regard to the cutaneous response to latex, the mean diameter of the papule decreased steadily with both of the commercial extracts tested: from 11 to 17 mm with the ALK-Abelló product, and from 45 to 18 mm with the Stallergènes.
extract. There were parallel decreases in the reactions to food allergens derived from banana, kiwi, and chestnut.

Absolute values of IgE to latex showed no significant alterations except at the end of the study, when an increase of approximately 4–6 URAST/ml was seen. However, IgE specific for chestnut clearly decreased during treatment from 0.72 to 0.30 kU/l. The behavior of IgG class specific for latex was similar to that of specific IgE. We did not detect latex-specific IgG4 during the study; however, this immunoglobulin apparently began to appear at the end of the study.

Clinical symptoms improved steadily, with an evident reduction in nasal obstruction and eye manifestations. This was corroborated by the patient, who reported improvement even in areas of the hospital that produced significant exposure to latex gloves, which she had previously been unable to tolerate. Acquisition of tolerance to the environment in her workplace was gradual during SIT, but was more pronounced once the maintenance period was started upon her discharge from the hospital and return to work. Because of the occupational nature of the allergy, the best provocation test for the allergen was constant exposure to latex in the workplace. However, we also used specific, controlled provocation tests. The patient entered a 1-m³ air-tight cabin and handled four pairs of latex gloves for 15 min; her clinical symptoms were then evaluated during the 6 h following this exposure to the allergen. Clinical examination after the provocation test showed that she had no cutaneous, eye, nasal, or bronchial symptoms during the following 6 h.

In terms of local reactions, tolerance of SIT was excellent, with no delayed local reactions and only one episode of immediate local symptoms during the following 6 h. This episode of immediate local symptoms during the following 6 h.

We consider SIT with latex to be highly effective, and found the allergenic extract used to be safe and well tolerated.

References

Nasal provocation with allergen induces a parallel release of ECP and matrix metalloproteinase-9 during the late-phase inflammatory response.

Allergen-induced matrix metalloproteinase-9 in nasal lavage fluid

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Key words: allergic rhinitis; eosinophil cationic protein (ECP); metalloproteinase; MMP-9.

• In an individual with allergic rhinitis, exposure to allergens leads to rapid release of mast-cell-derived mediators. In about half of the subjects, this immediate nasal response is followed 3–12 h later by a late-phase response [1]. This secondary response is induced by inflammatory cells, which have accumulated in response to mast-cell-derived chemotactic factors [1]. Bronchoalveolar eosinophilia is a hallmark of late-phase IgE-mediated reactions [2]. Okada et al. [3] recently showed that matrix metalloproteinase-9 (MMP-9) is required for migration of eosinophils through basement membrane components in vitro. Indeed, elevated levels of MMP-9 were recently found in the bronchoalveolar lavage fluid of asthmatics [4].

In a previous study [5], we measured albumin, eosinophil cationic protein (ECP), and MMP-9 in nasal lavage samples obtained before and up to 10 h after nasal allergen provocation. In the present study, we sought for MMP-9 in five series of these nasal lavage samples. Our results show a parallel release of ECP and MMP-9 after nasal provocation with allergen. Allergic rhinitis of the five patients involved in this study was confirmed by positive skin tests to grass pollen and/or house-dust-mite extract. Informed consent was obtained from all patients, and...