The prevalence of natural rubber latex (NRL) allergy has been estimated to be 5–18% in health care workers, and latex exposure has been one of the leading causes of occupational asthma in the last several years. We present the cases of two nurses who developed sensitivity to NRL, both with dermatologic symptoms and respiratory symptoms that included asthma. They were referred to the University of Maryland for evaluation of their allergies, then for occupational and environmental consults. The patients’ allergy to NRL was confirmed on the basis of clinical history, a positive skin test to latex, and the presence of latex-specific immunoglobulin E (IgE) serology by radioallergosorbent test (RAST). Both patients worked in the same community hospital for approximately 20 years; one was an endoscopy nurse and the other worked in the emergency department. Following the diagnosis of allergy to latex, both patients avoided direct skin contact with latex, but they continued to work in the same respective environments, where powdered latex gloves and other potentially sensitizing chemicals were used. Instead of improving, the clinical condition of the patients worsened and they remained symptomatic, even after they were removed from their workplace. Their airways reacted to low levels of a variety of sensitizers and irritants in the environment, and they became depressed. Both nurses were referred for vocational rehabilitation.

Case Presentation

Patient 1

A 49-year-old endoscopy nurse, who had worked in a community hospital for > 20 years, was diagnosed with allergy to latex after she experienced frequent peeling of her hands and local urticaria (hives) after skin contact with latex (e.g., gloves, condoms, dental procedures). Sensitivity to NRL was documented by positive radioallergosorbent test (RAST) at that time. The patient stopped wearing latex gloves but continued to work in the same hospital environment in the immediate vicinity of other hospital personnel who used powdered latex gloves and where latex balloons were brought in occasionally. Two years later, despite the avoidance of direct skin contact with latex products, she developed severe skin reactions with swelling and urticaria; she also developed upper and lower respiratory symptoms that included allergic rhinitis, hoarseness, wheezing, shortness of breath, chest tightness, and anaphylaxis. The symptoms were severe and led to several emergency department evaluations and treatments. She was transferred to an administrative area in the hospital but she remained symptomatic. The RAST for latex-specific IgE was repeated and was negative; however, skin tests for NRL were unequivocally positive. The patient developed allergic reactions to foods, and she started experiencing cough, wheezing, and shortness of breath when exposed to even low-levels of irritant chemicals. In addition, she had positive skin tests to common environmental allergens such as dust mites, pollen, and cockroaches, requiring desensitization with allergen immunotherapy. She developed laryngeal hyperactivity with laryngopharyngeal reflux and acid reflux disease. She was most comfortable in her home, which she converted to an “allergen free” environment. She became depressed due to her inability to work as a nurse and even to leave her home. She was released from work and referred for vocational rehabilitation a year later.

The patient was evaluated by several allergists and other specialists, including a gastroenterologist, a pulmonologist, a psychiatrist, a gynecologist, a psychologist, an otolaryngologist, and two occupational medicine physicians.

Despite the fact that the patient was removed from her work environment for > 3 years, her symptoms persisted and warranted several daily medications including a budesonide inhaler, a formoterol inhaler, loratidine, budesonide nasal spray, omeprazole, famotidine, venlafaxine, and albuterol (as needed). In addition, before leaving home and in order to prevent severe reactions if she encountered latex or other allergens and/or irritants, the patient often premedicated herself with cromolyn and ipratropium bromide inhalers and ipratropium bromide nasal spray. She also carried self-injectible epi-nephrine and oral diphenhydramine in the event of a severe allergic reaction.

The patient’s past medical history was significant for lactose intolerance, allergy to...
Using latex gloves, she continued to work in RAST. She became depressed because of the developed allergic reactions to several foods, to latex included skin symptoms with swelling. The second patient was a 46-year-old registered nurse who had worked for 19 years in the common model to wash her linoleum floor. The patient used unscented Tide laundry detergent (Procter & Gamble, Cincinnati, OH) to wash her clothes and Palmolive liquid detergent (Procter & Gamble, Cincinnati, OH) to wash her linoleum floor. The patient did not use spray chemicals to clean her work surfaces. After she was diagnosed with allergy to latex, the patient stopped using NRL gloves; however, her co-workers continued to use powdered latex gloves, and she continued to work with Cidex until she started experiencing breathing difficulty, and then she switched to isopropyl spray. At home, the patient implemented measures to make her environment “allergen free.” She removed existing carpets and rugs and she used a central vacuum which has a canister that is taken outdoors to clean. The house was equipped with central air conditioning, a heat pump, and special filters, and she had the ducts cleaned. No one smoked in the house. She washed the linens in hot water and covered her bed in material that is impermeable to dust mites, as advised by her allergist. She used unscented Tide laundry detergent (Procter & Gamble, Cincinnati, OH) to wash her clothes and Palmolive liquid soap (Colgate-Palmolive, New York, NY) to wash her linoleum floor. The patient did not use spray chemicals to clean her shower and tub; instead, she used hot water under pressure. She had no indoor plants and avoided going outside when the grass was being cut or when the lawn was being treated. Furthermore, the patient did not socialize in public places, except for a couple of restaurants where no latex gloves were used and smoking was prohibited.

**Patient 2**

The second patient was a 46-year-old registered nurse who had worked for 19 years in the emergency department of the same community hospital as patient 1. She had been diagnosed with contact dermatitis and mild intermittent asthma 5 years prior to the diagnosis of occupationally induced latex allergy. The diagnosis was based on her clinical history, positive NRL skin test, and seropositive latex specific IgE by RAST. The patient’s sensitivity to latex included skin symptoms with swelling and urticaria, rhinitis, intermittent wheezing, shortness of breath, and anaphylaxis. She also developed allergic reactions to several foods, documented by positive food-specific IgE by RAST. She became depressed because of the severity of her symptoms and their effects on her ability to work. Although she stopped using latex gloves, she continued to work in the emergency department for at least 1 year, where other personnel in the immediate vicinity used powdered latex gloves. Her symptoms did not improve; instead, they became severe and required that she discontinue direct patient care. She was reassigned to office work only. The patient’s condition worsened to the point that her respiratory symptoms usually recurred when she walked outside her house, which was the only place she felt comfortable and symptom-free. She was released from work and referred for vocational rehabilitation.

The patient’s past medical history was significant for seasonal and perennial allergic rhinitis. She reported that she had reactions to cats earlier in her childhood but became symptom-free as an adult.

**Discussion**

Two nurses, who worked at the same community hospital, developed severe latex allergy with dermatologic and respiratory symptoms, including urticaria and occupationally induced asthma and anaphylaxis. They also became sensitized to various chemicals and other environmental and food allergens. These nurses continued to experience respiratory symptoms that became severe, despite the fact that they were not using latex. Once the diagnosis of latex allergy was established, efforts were made to minimize the employees’ direct skin contact with latex, but little attention was given to their work environment. These sensitized workers remained in environments where powdered NRL gloves and other respiratory irritants and potential sensitizers (e.g., glutaraldehyde and other disinfectants) were commonly used and where latex balloons were brought in occasionally.

Occupational asthma caused by NRL has been reported in health care workers (Liss et al. 1997; Vandenplas et al. 1995). Most patients with latex allergy develop dermatologic symptoms as the first manifestation, with the most typical skin reaction being contact urticaria (Sussman et al. 1991). With continued exposure they may develop upper and lower respiratory symptoms, angioedema, and even anaphylaxis (Sussman et al. 1991). Allergic reactions to latex consist of immediate-type hypersensitivity reaction and delayed-type hypersensitivity reaction. The latter is a cell-mediated immune reaction in the skin that usually results from a hypersensitivity to one of the numerous chemicals added during processing (von Hintzenstern et al. 1991; Wyss et al. 1993), and it is rarely associated with systemic manifestations. However, patients with delayed-type hypersensitivity reactions are at greater risk of developing immediate reactions due to skin breakdown and resultant increased exposure to NRL (Charous et al. 1994; Turjaman et al. 1994). Immediate-type hypersensitivity reactions occur within minutes of exposure to latex products and are mediated by IgE to various latex proteins. Contact urticaria may develop in skin exposed to latex. If the latex proteins are aerosolized, wheezing, rhinitis, and conjunctivitis may occur. In severely allergic persons, reactions can progress to anaphylaxis. Delayed-type reactions and immediate-type reactions can also occur concurrently (Fuchs and Wahl 1992). The clinical manifestations of the present cases are consistent with immediate-type hypersensitivity that is IgE mediated.

The diagnosis of latex allergy is based on a comprehensive medical history and diagnostic tests. The skin-prick test is the preferred and most useful test in diagnosing type I latex hypersensitivity (Taylor and Praditsuwan 1996). However, there is a risk of causing anaphylaxis in highly allergic individuals (Kelly et al. 1993). The skin-prick test is the best predictor of latex allergy with 97% sensitivity and 100% specificity (Ebo et al. 1997), but the U.S. Food and Drug Administration (FDA) has not approved standardized latex solution to be used in the in vitro tests. A skin-prick test may show negative findings if the serum used did not contain the specific latex allergens responsible for the reaction in the individual being tested. Therefore, it is important that testing occur with more than one type of latex product, as well as with raw latex (Hamilton and Adkinson 1996).

The RAST identifies specific IgE antibodies to latex in the blood. Current FDA-approved in vitro latex IgE assays have lower sensitivity and specificity than the skin-prick test (Ebo et al. 1997) and produce a substantial number (25–28%) of false-negative and false-positive IgE antibody results (Hamilton et al. 1999). The presence of allergen-specific IgE does not always correlate with clinical symptoms (Bollinger et al. 2002). The RAST can confirm an NRL allergy diagnosis, but it should not be used as a screening tool because only 50% of a group of individuals identified as latex allergic by the skin-prick test had IgE antibodies to latex by the RAST (Taylor and Praditsuwan 1996).

In the two cases described above, we were able to document a history of clinical symptoms and positive skin-prick and RAST tests; we were therefore able to confirm the diagnosis of NRL allergy.

The existence of underlying atopy increases the risk of sensitization from workplace exposure to environmental allergens (Petsonk 2002). Health care workers who are atopic develop latex hypersensitivity more frequently than those who are not atopic (Bubak et al. 1992; Hunt et al. 1995). Taylor and Praditsuwan (1996) reviewed 44 patients with latex hypersensitivity and found that 77% of them were atopic; all but 2 of these patients were health care workers.
Subjects with latex allergy are more likely to develop sensitivity to other allergens, particularly foods. The prevalence rates of food allergy can be as high as 50% in latex-sensitive individuals, whereas the prevalence rate in the general population is 2% (Bezezhold et al. 1996; Blanco et al. 1994). Both of the nurses in our study had atopy, and they developed multiple food allergies.

Several studies (Heilman et al. 1996; Tarlo et al. 1994; Tomazic et al. 1994) have shown that the cornstarch powder used to lubricate gloves acts as a carrier for latex proteins; when the gloves are removed, latex proteins in dust particles become airborne and can be readily inhaled, even by those not wearing the gloves. Using powdered latex gloves can lead to measurable latex allergen in the environment on particles that are small enough to enter the airways and cause sensitization and symptoms (Swanson et al. 1994; Swanson and Ramalingam 2002).

Latex gloves contain various levels of latex allergens, depending on the brand and type of latex worn (Alenius et al. 1994). The amount of allergens aerosolized from the gloves correlates with the number of gloves used at the premises (Heilman et al. 1996; Sussman et al. 1998; Swanson et al. 1994). Latex aeroallergens are primarily generated by active glove use (Charous et al. 2000), and from balloons (Yunginger et al. 1994). High levels of latex aeroallergens have been detected in areas of heavy glove use, such as operating rooms, emergency rooms, and intensive care units (Heilman et al. 1996). Furthermore, airborne particles of powder and NRL proteins may remain suspended for up to 5 hr, contaminating the air and the ventilating system (Kelly et al. 1996). Therefore, for latex-sensitive health care workers with respiratory symptoms, the use of nonlatex gloves is only one of many steps required to reduce and eventually eliminate overall latex exposure.

A crucial step in the reduction or elimination of airborne NRL can be achieved by substituting nonlatex or powder-free NRL for powdered gloves. Such substitution has been found to be an effective prevention strategy that reduces the incidence of suspected latex allergy and specifically latex-related occupational asthma (Allmers et al. 2002; Tarlo et al. 2001; Vanderplas et al. 2002). After occupational exposure, the rates of sensitization and NRL-induced asthma rise dramatically in individuals using powdered NRL gloves but not in individuals using powder-free gloves (Charous et al. 2002). The Occupational Health Surveillance Program of the Massachusetts Department of Public Health (Boston, MA) conducted a survey of all acute care and several chronic care hospitals across the state. Of the hospitals with a program or policy to reduce employee exposure to latex, 40% reported a decrease in latex-related symptoms after the implementation of their program. Those hospitals with a program in place for >2 years were more likely to see decreases in symptoms than hospitals with a more recently established program. Most programs included the use of nonlatex or latex powder-free gloves (SENSOR 2002).

Some health care centers and professional offices have implemented a nonlatex glove policy throughout the workplace. Others have converted to powder-free latex in order to decrease the risk of airborne allergens. However, most of the health care settings, including dental offices, also use a number of detergents, germicides, and other chemicals that can also induce and exacerbate allergic conditions and play a role in the development of occupational asthma (Petsong 2002; Preller et al. 1996). For example, Cidex, a widely used cold sterilization solution for endoscopic equipment, is a well-known respiratory irritant and sensitizer (DiStefano et al. 1998; Petsong 2002). In a study that was based on 9 years of surveillance of work-related and occupational respiratory disease, McDonald et al. (2000) reported evidence for an increase in cases of occupational asthma due to latex and glutaraldehyde. The prevalence of work-related lower respiratory tract symptoms in hospital endoscopy nurses has been reported to be 8.5% and 66.6% in current employees and ex-employees, respectively (Vyas et al. 2000).

It is likely that patient 1’s continuous exposure to Cidex, even after she was diagnosed with occupational asthma, contributed to her persistent clinical symptoms and her developing reactions to all sorts of environmental irritants.

In long-term follow-up studies of workers with sensitizer-induced occupational asthma, the clinical symptoms have been reported to persist in approximately 70% of affected workers who were no longer exposed to the sensitizing agent (Chan-Yeung and Malo 1999; Malo et al. 1992). It has been postulated that once a person is sensitized to an allergen or chemical, he or she may develop asthma, which may be “driven and maintained by the persistence of a specialized subset of chronically activated T-memory cells sensitized against an array of allergenic, occupational, or viral antigens” (Kay 1997). This can explain the fact that asthma can be aggravated in a nonspecific manner by exposure to dust, smoke, fumes, and low levels of irritant chemicals. Persistence of exposure to the sensitizing agent leads to worsening of asthma (Chan-Yeung and Malo 1999), especially in health care workers with latex allergy (Charous et al. 1994), and early removal has been consistently found to be associated with a better outcome.

To establish a latex-safer environment, an effective program or policy would include provision of screening for and surveillance of workers at risk of becoming sensitized to latex. Such screening should be based not only on a positive latex-specific IgE test but also on a clinical history elicited through a questionnaire. Indeed, such a program was implemented at the University of Maryland Hospital in 1997 (Bollinger et al. 2002). A latex sensitization rate of 8.2% was found in the subjects screened. Of note, 45% of the latex IgE positive employees were asymptomatic and 21% of the latex specific IgE negative employees were symptomatic with latex exposure. All employees with either latex-associated symptoms and/or latex-specific IgE were advised to avoid latex products. The hospital gradually converted all gloves to powder-free low protein. Since the program’s inception, there have been no further latex-related workers’ compensation claims. Similar decreases in latex-related workers’ compensation claims have been described by Tarlo et al. (1994, 2001) in Canada after conversion to powder-free gloves.

Conclusion

In the present report we describe the cases of two health care professionals who developed allergy to NRL, persistent occupational asthma, and subsequent reactions to a variety of environmental sensitizers and irritants. As with their cases, when latex allergy is diagnosed, special attention should be given to the patient’s work and home environments, because timely control of exposure is important to minimize further damage and long-term adverse effects. Health care providers must be aware of the limitations of the RAST test and the importance of clinical history in screening for or diagnosing latex allergy. Finally, education of personnel about latex allergy, appropriate handling of potentially harmful chemicals, and immediate reporting of adverse health effects is crucial to maintain a healthy and safe work environment.

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