Chapter 29

Dealing with Latex Allergies

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Background

Documentation of natural rubber latex (NRL) allergy among healthcare workers was first noted in 1984.[1] Although earlier reports do exist, this was the first to document anaphylactic reactions among healthcare workers to surgical gloves. Latex allergy has, therefore, been coined as a "new disease." The prevalence of this allergy has reached epidemic proportions in highly exposed populations since 1983. Estimates of the incidence of latex allergy throughout the general population range from 1–6%, while certain pediatric populations range from 18–73%.[2-4] Among healthcare workers, the projected incidence of NRL allergy is between 8% and 17%.[5-7] There has been a corresponding increase in public awareness of latex allergy. The incidence of latex allergy varies according to skin test methods used, small sample populations, and the use of selected populations.[7,8]

History

Latex Origins and Uses

The oldest rubber artifacts have been found in Veracruz, Mexico, and were carbon dated back to 1600 B.C.[9] These early Mesoamerican artifacts were made from latex derived from the *Castilla elastica* tree. *Castilla* latex was too brittle to retain its shape and had to be processed with juice from a morning glory vine to create a more malleable and elastic product.[9]

Today, nearly all commercial latex is a natural product derived from the latex sap of *Hevea brasiliensis* tree. The latex derived from this tree contains rubber particles, NRL, which is widely used in the manufacturing of medical devices as well as everyday articles.

Note: The author acknowledges Philip Schneider who authored this chapter in the first edition of this text.

When processed, this NRL has highly desirable properties.[1]

Source of Latex Allergens

The two basic manufacturing types of NRL products are *molded* and *dipped*. Molded rubber products are comprised of dried rubber and coagulated latex. These products are made rigid through compression. Items falling into this category include drug vial stoppers, syringe plungers, and intravascular injection ports used in virtually all healthcare settings.

Dipped rubber products are manufactured through a process of dipping a mold into NRL. Examples of these types of products include surgical gloves, coatings for tool handles, and condoms.

The distinction between these two types of manufacturing processes is critical to antigenicity. Molded rubber products do not leach antigens to any appreciable extent, but dipped rubber products leach antigens in abundance.[10,11] Dipped rubber products (e.g., surgical gloves) are often manufactured with a dry lubricant such as corn starch, which also serves as a potent vehicle for the aerosolization of NRL antigens. NRL antigens are ubiquitous in nature and can be detected in NRL products, foods, spices, pollen and plants. This ubiquity raises the possibility that sensitization can occur from multiple sources and may be difficult to determine in individual patients. Certain fruits—such as bananas, chestnuts, kiwi, avocado, and tomato—show cross-reactivity, presumably due to resemblance to a latex protein component.[10] Hence, the term *latex-fruit syndrome* has emerged.[12] Sensitization may, therefore, actually occur from nonrubber products.

Allergic Reactions

People with allergies experience hypersensitive immune system responses to substances called *allergens*. Common allergens include dust, pollen, and pet dander. Allergens are a type of antigen, which is any foreign substance that triggers an immune response. These allergens include substances that are innocuous to nonallergic subjects as well as generally harmful such as viruses and bacteria. Allergic reactions have been classified as Types I, II, III, or IV. Each type has unique characteristics and time of onset. Allergic reactions can involve most of the components of the immune system including cellular, immunoglobulins, complement, and cytokines.

Type I reactions (IgE mediated; also called immediate and anaphylactic reactions)—These reactions generally occur within 30 minutes of exposure to the antigen (e.g., protein in NRL) and are the most severe reaction. After initial exposure, allergen from NRL binds to immunoglobulin E on the basophils or mast cells. During this initial exposure to the NRL antigen, predisposed individuals produce IgE specific for this antigen. After subsequent exposure to the antigen, an IgE-antigen complex is formed and binds to the basophil or mast cell. This binding results in the release of inflammatory mediators such as histamine, serotonin, chemotactic factors, bradykinin, etc. Systemic and anaphylactic reactions to NRL are mediated through Type I allergic reactions. This most severe form of latex allergy is often referred to as *true latex allergy*. Individuals experiencing this type of reaction have a systemic response to latex that may result in fulminant anaphylaxis.[13]

Type II or cytotoxic reactions—These reactions are characterized by cell destruction (usually blood elements) secondary to cell-associated antigen initiated cytolysis and usually occurs within 5–12 hours. Type II allergic reactions are mediated by IgG or IgM.

Type III or immune complex reactions—These reactions are caused by antigen-antibody complexes that deposit on blood vessel walls and activate complement resulting in local or disseminated inflammatory reactions. Type III allergic reactions typically manifest in 3–8 hours.

Type IV or cell-mediated or delayed hypersensitivity reactions—These reactions occur within 24–48 hours and are caused by antigen activation of lymphocytes (T cells), which in turn release inflammatory mediators. Clinical hallmarks include erythema, scaling, and vesiculation.[14] This is generally an allergic contact dermatitis and is much more common among latex sensitive individuals than Type I reactions and is not life threatening (**Table 29-1**).[15]