Contact Urticaria

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Contact urticaria is a wheal reaction that appears, usually repetitively, within minutes or up to one hour after contact with a causative agent [1–3]. The wheal reaction generally disappears within a few hours but it can sometimes evolve to generalized urticaria and even anaphylaxis [3, 4]. The wheal reaction may be allergic (immunologic contact urticaria) or non allergic (non immunologic contact urticaria) . Some substances can provoke contact urticaria, acting on intact skin, while others induce the complaint on already damaged or eczematous skin [4–6].

21.1 Non Immunologic Contact Urticaria

Non immunologic contact urticaria is the most prevalent type of contact urticaria [7, 8], caused by a wide variety of agents. It occurs without previous sensitization in nearly all exposed individuals [2]. Skin lesions are generally restricted to the site of contact, and systemic manifestations are rarely observed [1]. The severity of the urticaria will depend on the amount of urticant agent, the concentration, and exposure time [9].

Examples of causal agents include animals (e.g., arthropods, caterpillars, corals); foods (pepper, mustard, thyme); fragrances and flavorings (e.g., balsam of Peru, cinnamic acid, cinnamic aldehyde); medicaments (e.g., benzocaine, camphor, witch hazel); metals (cobalt); plants (nettles, seaweed); and preservatives and disinfectants (e.g., benzoic acid, formaldehyde) [10].

21.2 Immunologic Contact Urticaria

Immunologic contact urticaria involves a type 1 hypersensitivity reaction mediated by allergen-specific immunoglobulin E (IgE) and, therefore, requires a prior sensitization phase [11–13]. Prior sensitization can occur through contact or exposure of the skin, mucous membranes, respiratory tract, or gastrointestinal tract. Two types of agents can cause immunologic contact urticaria [14], namely proteins, such as natural rubber latex, with a high molecular weight that is often more than 10,000 kDa, and hapten chemicals, which conjugate with carrier proteins (e.g., albumen): the hapten-carrier

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Table 21.1 Stages of progression in contact urticaria

Stage	Description
1	Localized reaction (redness and swelling) with non specific symptoms (burning, itching, tingling)
2	Generalized reaction
3	Extracutaneous symptoms (rhinoconjunctivitis, orolaryngeal and gastrointestinal dysfunction)
4	Anaphylactic shock

protein can induce sensitization [8, 13, 14]. Pre-existing conditions, such as atopic dermatitis, may favor this condition [8, 13–15]. Generalized reactions and/or extracutaneous reactions are frequent, and are denominated contact urticaria syndrome [5]. In Table 21.1, the four stages of progression in contact urticaria syndrome are described [16, 17].

21.3 Contact Urticaria of Unclear Mechanism

There is an additional type of contact urticaria which comprises reactions with mixed features of both immunologic and non immunologic mechanisms, whose mechanisms and pathophysiological features are not well understood [1, 5, 7, 16]. A well-known example is the contact urticaria due to oxidizing chemical ammonium persulfate (contained in hair bleaching products) [18].

21.4 Occupational Contact Urticaria

Occupational contact urticaria can be immunologic or non immunologic; it accounts for 1–8% of occupational skin disorders [15]. Immunologic contact urticaria to natural rubber latex is particularly frequent among health care personnel, but contact urticaria to a wide variety of other substances occurs in many occupations [19]. Among those at high risk are cooks, bakers, butchers, restaurant personnel, veterinarians, seafood handlers (fishermen), laboratory technicians, hairdressers, florists, gardeners, and forestry workers [8, 9, 11, 20, 21].

Occupational contact urticaria has been described due to cyclic acid anhydrides in welders, painters, plumbers, chimney sweeps, packers, and electricians [22, 23]. The risk of sensitization against all proteins is high in the presence of atopy in occupational contact urticaria [13, 16].

21.5 Triggers of Contact Urticaria

21.5.1 Cosmetics

Cosmetic components can cause contact urticaria with or without systemic symptoms [24]. This problem is probably grossly underdiagnosed because patients fail to report the reactions and just discontinue the use of the product.

Hair Dyes and Hair Bleaching. Hair dye chemicals such as *p*-phenylenediamine and its derivatives, such as *p*-aminophenol and *p*-methylaminophenol [25], and toluene-2,5-diamine [26] can cause contact urticaria. The reactions seem to occur only after oxidation by H_2O_2, and are attenuated when the antioxidant sodium sulfite is added to the mix [26]. Aside from paraphenylenediamine, reactions to Basic Blue 99 (a mixture of 23–32 substances at various concentrations and with varying compositions), Basic Brown 17 (an azo dye), and other reactive dyes have also been reported to cause contact urticaria, mainly provoked by occupational exposure [27, 28]. Ammonium persulfate and potassium persulfate, used for hair bleaching, can also cause the affliction through a mechanism that is still unclear [29–33]. Hairdressers exposed to these products on a daily basis are at risk of developing cutaneous reactions [34, 35].

Fragrances. Fragrances have been reported to cause both immediate and delayed

hypersensitivity reactions. A multicenter study in Hungary found that 6.1% of patients with contact dermatitis to fragrances also reported an immediate contact urticaria reaction [36]. Cinnamal is the allergen most frequently reported to induce the dermatitis [24].

Sunscreens. Contact urticaria to sunscreens is rare but has been seen with benzophenone-3 (INCI; syn. 2-hydroxy 4-methoxy benzophenone, oxybenzone), a common ultraviolet (UV) A/UVB sunscreen [24, 37]. The severity of the clinical reaction depends partly on the area of exposed skin so patch testing does not necessarily elicit anaphylaxis. Contact urticaria can occur from exposure to hydrolyzed wheat protein in cosmetic creams and shampoos [38]. Three patients reported reactions to a hair conditioner containing hydrolyzed wheat protein, one on the hands while the other two developed acute urticaria on the head and neck. All were atopic patients [39].

21.5.2 Latex

Latex is probably the most important cause of contact urticaria [40], especially among medical and orthodontic staff [1, 7]. Although the incidence of latex allergy has declined in recent years, it is still a major health care issue. Latex is a milky fluid consisting of the cell cytoplasm of the tree *Hevea brasiliensis*; the cell nucleus and mitochondria are not expelled during harvesting, thereby allowing cell regeneration to occur [41]. Latex has four main components, namely rubber particles, lutoids, Frey Wyssling particles and the cytosol. The rubber particles are the most numerous organules of lactiferous cells. They consist of spherical drops of cis-1,4-polyisopropene enwrapped by a thin layer of phospholipoproteins [42]. Two proteins that synthesize cis-1-4-polyisopropene have been identified: the first is cis-prenyltransferase (38 kDa), a hydrophobic enzyme that catalyzes the addition of isopropene units until a polyisopropene chain several thousand units long has been formed. The second, the "rubber elongation factor", is a stabilizing cofactor (14.6 kDa) necessary to ensure the efficient function of the cis-prenyltransferase [43]. Lutoids are vacuoles that account for 10–20% of the latex volume, and are important for its coagulation. Heveine (4.7 kDa) and proheveine (20 kDa) are the main proteins of lutoid bodies. Heveine accounts for 70% of the lutoid proteins and its structure is homologous to that of various agglutinins of plants, such as rice, potato, and grain. Frey Wyssling particles (2–3% of the latex volume) play a biological role that has not yet been clarified. The cytosol makes up 40–50% of the volume; it contains carbohydrates, organic acids, amino acids, nucleotides and proteins that are important in the synthesis of isoprene.

The prevalence of latex allergy depends on the population studied, spanning a wide range from 3 to 64%; latex sensitization in the general population ranges from 5.4 to 7.6% [44]. A risk factor is repeated contact with, or prolonged exposure to, latex-containing products especially in the medical setting. It has been calculated that approximately 10–20% of health care workers are sensitized to latex [45] but contact with other types of latex-containing articles both in medical and non medical settings may also have a role. Workers in the latex manufacturing industry are another subpopulation at risk [46], as are food handlers, domestic workers, florists, gardeners, and hairdressers [46–50]. Other risk factors for allergy to latex include preexisting skin injuries, atopy, spina bifida, and certain genetic profiles (HLA-DR phenotypes) [51]. Preexisting skin injuries such as hand dermatitis alter the skin barrier and can lead to increased penetration of latex proteins [52, 53]. Atopic individuals have an enhanced propensity to produce latex-specific IgE and are at risk of developing a latex allergy [54, 55]. Spina bifida patients have a high risk of latex sensitization due to the frequent number of surgical procedures early in life [56, 57].

Immunologic contact urticaria from latex is a type I IgE-mediated hypersensitivity reaction, and is the most frequent form of presentation of latex allergy [58]. It typically occurs within minutes of latex exposure. Symptoms may be mild, with urticarial reactions, rhinoconjunctivitis, or mucosal swelling. More severe systemic

symptoms may develop, including generalized urticaria, asthma, bronchospasm, hypotension, and anaphylactic shock [59–62]. Latex allergy is the second main cause of intraoperative anaphylaxis (after muscle relaxants) and is the first cause of anaphylaxis in children [58, 63–66]. Reactions to latex usually occur during the maintenance phase of the operation, whereas when anaphylaxis is caused by opiates or muscle relaxants, it is usually during the induction phase. Several factors may influence the severity of reactions, such as the route of exposure (e.g., skin, mucosa, intravascular), source of exposure (gloves vs other exposure), latex type (ammoniated vs non-ammoniated), and individual immune responses [67]. Adverse reactions may also result from inhalation of airborne allergens bound to substances such as glove powder [68, 69]. Airborne latex allergy most commonly manifests as rhinoconjunctivitis but can also trigger asthma and contact urticaria [60, 70]. Fifteen different allergenic proteins have been identified and registered by the International Nomenclature Committee of Allergens [71]. Hev b1, 2, 3, 4, 5, 6, 6.01, 6.02, 7.01, 13, and 14 have been identified as the most sensitizing *Hevea* allergens [72]. Additional allergens continue to be investigated. A few studies have suggested that different latex allergens could sensitize different categories of individuals [73]. Natural rubber latex Hev b 1 and Hev b 3 are the major protein allergens involved in patients with spina bifida [73]. Hev b 2 and Hev b 4 may play a more important role in health care workes with latex allergy [74]. Hev b 5 is a major allergen in the majority of both health care workers and children with latex allergies [75]. Although some latex allergens, such as Hev b 1 and Hev b 6, may be specific for latex, other latex allergens have been found to share IgE epitopes with plant-derived foods. This implies that sensitivity to latex may be triggered due to sensitization to homologous allergens in certain foods, and vice versa. The latex-fruit syndrome (or "latex food allergy syndrome") is due to this cross-reactivity of latex proteins to similar proteins in fruits and vegetables [76]. The most common foods implicated are bananas [77], avocado [77, 78], chestnuts [77], and kiwi [79]. Less commonly reported are papaya, lychee, fig, peach, potato, chickpea, spinach, and the leafy green vegetable phuk waan-ban [41, 72, 80].

21.5.3 Topical Medicaments

Immunologic contact urticaria may occur due to the active agent or the preservative, base, or additives. Antibiotics can induce the dermatitis, often associated with anaphylactic reactions. Antibiotics reported as causes of contact urticarial include bacitracin, cephalosporin, chloramphenicol, gentamycin, neomycin, penicillin, rifampicin, and streptomycin [81]. Topical local anesthetics can also induce contact urticaria [82], but most cases of contact urticaria to local anesthetic agents are non immunologic [83]. Nitrogen mustard used to treat mycosis fungoides was associated with contact urticaria with an anaphylactoid reaction in one case [84].

21.5.4 Foods

Virtually any food is capable of eliciting an immunologic contact urticaria response [85]. Table 21.2 lists foods that have been reported as a cause of contact urticaria. Contact urticaria from food is usually observed in an occupational setting and the foods most frequently responsible are apple, potato, carrot, and tomato; shellfish and seafood such as prawn and lobster are also sources [86–88]. Food handlers affected by immunologic contact urticaria to raw seafood can usually tolerate eating cooked seafood provided that the seafood is protein denatured by cooking [88]. Wheat allergens can provoke asthma and contact urticaria among bakers [89]. Cross-reactivity between pollens and fruits (Table 21.3) is responsible for a mucosal immunologic contact urticaria [90]. Contact hypersensitivity syndrome (also known as oral allergy syndrome, OAS), is a form of contact allergy reaction that occurs upon contact of the mouth and throat with raw fruits or vegetables. The most frequent symptoms include itchiness

Table 21.2 Foods as a cause of contact urticaria

Vegetables
Asparagus Beans Cabbage Celery Fungi Garlic Lettuce Mushroom Mustard Onion Rice Soybean Tomato
Fruit
Apple Apricot Banana Kiwi Lemon Lime Mango Orange Peach Peanut Plum Strawberry Watermelon
Meat: beef, calf, lamb, chicken **Fish**: cod, crab, frog, seafood, raw fish **Other animal products**: cheese, egg, honey, milk

Table 21.3 Common cross-reactions between pollen/plant allergens and fruit

Pollen/plant	Common fruit
Birch	Apple, pear, carrot, celery, tomato, cherry
Mugwort	Carrot, celery, aniseed, peach
Ragweed	Melon
Goosefoot	Banana, melon, peach
Latex	Avocado, banana, chestnut, kiwi, mango, melon, papaya, tomato

or swelling of the mouth, face, lips, tongue and throat. Symptoms usually appear immediately after eating raw fruits or vegetables, although in rare cases, the reaction can occur more than an hour later. Rarely, the affliction can cause severe throat swelling leading to difficulty in swallowing or breathing. Gastrointestinal symptoms, such as diarrhea and stomach-ache, are uncommon. Some rare cases of life-threatening reactions, with angioedema or shock, have been reported. Cooked food, with the exception of nuts and celery, is generally safe. Sometimes the affliction can be associated to an exacerbation of hay fever symptoms. Handling the fruit can also cause contact urticaria.

21.5.5 Plants

Exposure to several plants can cause contact urticaria, especially in the occupational setting. Common causes of contact urticaria are Compositae, ivy yucca, spathe flower, Chinese rose [14]. Christmas cactus, Barberton daisy, and Madagascar jasmine have also been reported as causes of contact urticaria [91]. Profilin, present in several plant species, has been suggested as a common causative agent for immunologic contact urticaria [11]. Chamomile tea, a folk remedy used to treat conjunctivitis and other ocular reactions can induce immunologic contact urticaria, presenting with eyelid angioedema, in patients sensitized to Compositae and especially to Artemisia [92, 93].

21.5.6 Animal-Derived Proteins

Animal derivatives such as animal hair and secretions can induce immunologic contact urticaria in animal handlers, farmers and veterinarians. In Finland, the dermatitis to cow dander is very frequent because cows are kept indoors most of the year so dander exposure is increased [14]. Dog and rat saliva, animal hair, cow placenta, dog milk [94], rat tails, and guinea pigs can all be causative agents in subjects handling animals [81]. Also animal-derived protein allergens in cosmetics have been reported among the causes, such as fish-derived elastin-containing cosmetics [95], while lactalbumin from a mare's milk-containing cosmetic cream has also been reported [96]. Niinimäki and Coll. observed 11 hairdressers with hand dermatitis found to be sensitized to Crotein Q® (hydroxy propyl trimonium hydrolysed collagen) [38]. Prick test reactions to very low concentrations of this substance and specific IgE antibodies against Crotein Q® were elicited [38].

21.5.7 Textiles

Silk, wool, rubber and nylon may produce immunologic contact urticaria [97]. Silk has often been reported as a cause of immediate-type reactions (immunologic contact urticaria, anaphylaxis and respiratory disease) [97–99], and might be an even more frequent finding in atopic subjects [98, 99]. Silk allergens include the silk fiber itself, the gum or glue (sericin) contained in raw silk and the silkworm or insects of the genus *Anthrenus* contained in silk materials [98, 99]. Asthma, rhinitis, anaphylaxis, and eczema may or may not accompany the urticarial reaction [97].

21.6 Diagnosis

The diagnosis of contact urticaria involves detailed clinical history taking, clinical examination, skin test and specific IgE measurement. After a thorough history has been taken, the physician should proceed with a focused physical examination, checking that antihistamines have not been used within two days of performing the examination. Testing commonly employs a step-wise approach and may include the open test, prick test, scratch test, and use test, making sure to include positive and negative controls during each step. The first step in diagnostic testing for immediate IgE-mediated allergy is an open test: [100] it is usually performed on the ventral forearm using 0.1 mL of the suspected urticarial substance and spreading it over an area measuring 3 × 3 cm. Saline is used as negative control. The open test is first performed on non affected skin and if negative, on slightly affected or previously affected skin [4, 5]. When performing an open test, physicians should take precautions against anaphylaxis. If the open testing is negative, prick testing is usually performed next in the diagnosis of contact urticaria, and is considered the diagnostic method of choice if open testing is negative [101]. Prick testing is generally considered safe, but isolated cases of anaphylaxis have been reported [102].

The test substance is applied to the volar aspect of the forearm and pierced into the skin using a lancet. Reading of a prick test is usually performed after 30 minutes. A scratch test is more useful for non-standardized allergens [3]. The area of the skin is scratched with needles after the allergen has been applied. Reading of this test is done after 30 minutes. If skin tests are negative, the use test is performed with the incriminated agent. For example, a person with latex-induced contact urticaria would wear latex gloves during testing.

RAST for allergen-specific IgE are not available for all agents responsible for contact urticaria [14]. RAST for allergen-specific IgE to latex is highly positive in sensitized patients, but a negative RAST test does not exclude the diagnosis of immunologic contact urticaria.

21.7 Therapy

The most important intervention in sensitized subjects is to ensure the complete avoidance of the offending antigen, to prevent recurrent symptoms and possibly life-threatening anaphylaxis. It is recommended that patients should always have injectable epinephrine and antihistamines on hand with them [3]. They could be required to treat a life-threatening reaction. Patients who develop contact urticaria to latex need to take care to avoid this specific substance in the future. Allergen immunotherapy may be an effective option in treating latex-allergic patients [103].

21.8 Prevention

In the occupational setting contact urticaria may be prevented by applying preventive measures, that consist in the elimination, by substitution, of the occupational contact allergen and the use of personal protective equipment. Powdered latex gloves should particularly be avoided as the culprit antigen may become aerosolized. In fact, elimination of powdered latex gloves may

be the single most effective measure in the overall risk reduction of latex sensitization and clinical reactions.

As underlined above, the most important intervention for secondary prevention is complete avoidance of the offending antigen, to guard against recurrent symptoms and the risk of life-threatening anaphylaxis.

People with a latex allergy should be aware of all other products besides gloves that contain latex both in the hospital and the home setting. These products include (in the hospital) catheter stoppers, elastic bandages, tourniquets tubes, and masks. In the domestic setting, they include balloons, condoms, mats, bottles, and baby bottle nipples. Alternatives to latex are available and include nitrile, neoprene, and polyvinyl chloride. Nitrile provides a similar protection against infection to that offered by latex; synthetic polymers, such as neoprene, can be used as an alternative in surgical procedures [58, 104, 105].

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