



Anaphylaxis as occupational risk

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Purpose of review

Anaphylaxis is a severe form of allergic reaction that may cause death. Main triggers of anaphylaxis have been reported to also cause occupational anaphylaxis (OcAn). The purpose of this article was to summarize the current scientific evidence on OcAn and review the specific literature published in 2012 and 2013.

Recent findings

Allergens derived from *Hymenoptera* and natural rubber latex are the most frequently reported triggers of OcAn, but other high and low-molecular weight agents have been described. Among the latter, foods, insects, snakes, chemicals, and medications have been described in the last 2 years. Furthermore, reviews on the clinical significance of immunological contact urticaria as a risk factor for systemic allergic reaction and on *Hymenoptera* venom immunotherapy have been published.

Summary

OcAn is a serious event, and several agents and occupations have been identified as responsible. In any occupational setting, where there is a worker with a history of previous anaphylactic reactions, and in occupations at greater risk, a written emergency management plan of anaphylaxis episodes, along with the availability of adrenaline and trained personnel, are mandatory. Venom immunotherapy should be considered for patients with *Hymenoptera* venom allergy.

Keywords

hymenoptera allergy, latex allergy, occupational anaphylaxis, triggers of anaphylaxis, work-related anaphylaxis

INTRODUCTION

Anaphylaxis is a severe form of allergic reaction that is rapid onset and if left untreated may cause death [1]. Several agents used at the workplace may induce anaphylaxis; a series of case reports have been reported in the literature, but no systematic review is available. The most common triggers of anaphylaxis at the workplace are *Hymenoptera* stings and natural rubber latex (NRL). Other triggers less frequently reported are medications, food, animal/insect bites, and chemicals [2,3].

The purpose of this article was to summarize the current scientific evidence on occupational anaphylaxis (OcAn) and review the specific literature published in 2012 and 2013.

DATA SOURCES, SEARCH STRATEGY, AND STUDY SELECTION

The literature was reviewed by the authors using Medline search. In the selection of articles, we followed the reported definition of anaphylaxis

mentioned earlier [1]. Published studies in English were identified from PubMed from January 1, 2012 to November 23, 2013. We used the following keywords search strategy (work* OR occupation*) AND (anaphylaxis OR anaphylactic OR anaphylactoid OR 'venom allergy' OR 'systemic reaction*').

Reference citations published within the 2012–2013 time frame that had not been otherwise identified in the initial search were added wherever relevant.

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Curr Opin Allergy Clin Immunol 2014, 14:328–333

DOI:10.1097/ACI.0000000000000066

KEY POINTS

- *Hymenoptera* venom and latex allergens are the most common triggers of OcAn, but several occupations may be at risk.
- The diagnosis of OcAn requires confirmation of a diagnosis of anaphylaxis and identification of the culprit agent.
- The management of OcAn should follow current guidelines on anaphylaxis; in beekeepers, immunotherapy should be considered.
- Workers with a diagnosis of OcAn should carry two preloaded adrenaline delivery systems and be trained for use. Any further exposure should be avoided.
- A written emergency management plan, adrenaline, and trained personnel should be available in every workplace.

OCCUPATIONAL ANAPHYLAXIS: CURRENT DEFINITION AND KEY TRIGGERS

There is no universal agreement upon definition of OcAn. We propose to define OcAn as ‘anaphylaxis arising out of causes and conditions attributable to a particular work environment’.

The triggers of OcAn may come into contact with the individual by inhalation or skin contact (mainly, in a person with preexisting skin disease), by injection through stings or bites, or following hand-to-mouth transmission in workplaces with poor industrial hygiene practices [3].

Any workplace agent capable of causing occupational asthma or generalized urticaria may theoretically cause anaphylaxis [4]. Both high-molecular weight (HMW) and low-molecular weight (LMW) agents have been described as cause of OcAn (Table 1) [2,3]. HMW allergens derived from *Hymenoptera* venom and NRL are the most frequently reported.

MECHANISMS AND CLINICAL PRESENTATIONS OF OCCUPATIONAL ANAPHYLAXIS

Underlying mechanisms of anaphylaxis and of OcAn may be allergic, IgE-mediated or non-IgE mediated, or nonallergic. In non-IgE-mediated reactions, symptoms may be similar to IgE-mediated reactions; however, an IgE-mediated mechanism is not demonstrable.

Anaphylaxis usually occurs minutes after exposure to the allergen, but in rare cases, the latency period between exposure and reaction may increase

Table 1. Triggers of occupational anaphylaxis reported in the literature and related occupations

Trigger	Occupations
Drugs	Pharmaceutical plants; healthcare settings (preparation of medication)
Food-derived proteins	Food-processing plants
<i>Hymenoptera</i> and other insect bites/stings	Beekeepers, greenhouse, and bumblebee farm workers; agriculture, parks and forestry, and gardening and landscaping workers; gardeners, construction workers, and car drivers; aquarists
Animal bites	Laboratory animal workers; snake farm workers; workers with vipers
NRL	Healthcare workers; manufacturing plants with NRL in the production process; other jobs wearing NRL gloves
Chemicals	Chemical plants; agriculture (insecticides and fungicides); hair dressing (ammonium persulfate, dyes), welding (chromium, cobalt); healthcare settings (disinfectants)

NRL, natural rubber latex. Adapted from [3].

up to several hours [1]. The clinical pattern of symptoms of OcAn depends on the route and frequency of exposure (repeated exposure may lead to progressive allergic reactions or, by contrast, to clinical tolerance, as described in beekeepers receiving more than 200 stings per year who tolerated bee stings without systemic hypersensitivity reactions [5]); type of allergen involved [patients and healthcare workers (HCW) exhibit a different pattern of IgE sensitization to NRL compounds, being Hev b 6.01/Hev b 6.02 the polypeptides more frequently involved in OcAn] [6]; other cofactors, such as exercise during work, may be a trigger of exercise-induced anaphylaxis.

When anaphylaxis occurs in the workplace, it is highly likely to be caused by workplace exposures. However, allergic manifestations also because of nonoccupational exposures, for example, ingestion related, may manifest at work; therefore, before labeling a reaction as OcAn, an in-depth investigation is required.

Moreover, workers who have developed sensitization to an allergen in the occupational setting may only experience anaphylactic reactions in non-workplace settings (e.g. HCW with NRL allergy who may complain symptoms when undergoing medical/surgical procedures or workers ingesting food or medication handled at the workplace). Conversely, a reaction occurring in the workplace may also be the result of a cross-reaction between a nonoccupational allergen, to which the patient is sensitized, and an occupational allergen.

DIAGNOSIS OF OCCUPATIONAL ANAPHYLAXIS

The diagnosis of OcAn requires confirmation of the diagnosis of anaphylaxis (Table 2) [1] and identification of the occupational agent inducing the reaction. A careful clinical and occupational history, including exposure documentation; investigation of concomitant individual risk factors, such as high levels of basal tryptase (possible mastocytosis), diagnoses of asthma, and/or cardiovascular disease, as well as concurrent medications, such as β -blockers and angiotensin-converting-enzyme inhibitors; skin prick tests or intradermal tests (performed under controlled conditions at least 3–4 weeks after the event); and/or serum-specific IgE assessment for occupational allergens are the key steps of the diagnostic workup [3].

OCCUPATIONAL ANAPHYLAXIS DUE TO MEDICATIONS AND FOODS

OcAn due to drugs and foods, which are the main triggers of anaphylaxis, has been only occasionally described. It may be supposed that exposure to drugs or foods by inhalation or direct skin contact, as occurs in the workplace, does not easily lead to anaphylaxis as ingestion does.

Medications

The most common class of drugs associated with OcAn is beta-lactam antibiotics. In the past 2 years, few articles have shown work-related sensitization, systemic reaction, and anaphylaxis due to medications. In a study on 161 Korean HCW, Kim *et al.* [7] described sensitization to cephalosporins in 17.4% of workers, with specific IgE to cefotiam,

ceftriazone, or ceftizoxime, and anaphylaxis due to cephalosporins in one nurse.

Immunotherapy has also been identified as causing OcAn. Bandino and Tankersley [8] reported a case of a 32-year-old female technician who had anaphylaxis after a needlestick while preparing immunotherapy vials of timothy grass extract [8]. Previously, she had allergic rhinitis with sensitization to timothy grass. A few minutes after the needlestick, she had a local wheal-and-flare response followed by rhinoconjunctivitis, diffuse pruritic flushing and urticaria, and angioedema of the face, periorbital region, and tongue. Her initial blood pressure was 102/50 mmHg.

In an article aimed to explore the knowledge, attitudes, and beliefs of nurses who administered chemotherapy to children, Gibson *et al.* [9] found that nurses were worried about dealing with allergic/anaphylactic reactions to drugs, although no anaphylactic reactions due to chemotherapy were reported.

Foods

Occupational exposure to foods more often leads to contact dermatitis, urticaria, and respiratory symptoms. In 2012, Ismail and Maibach [10] published a review on the clinical significance of immunological contact urticaria to processed grains, a type I hypersensitivity reaction with potential complications including organ system involvement other than skin and even anaphylaxis and death. A vast majority of immunological contact urticaria is work related [11]. An important message of that review was that contact dermatitis or other disease disrupting skin permeability should be regarded as a risk factor for systemic allergic reaction [10]. Several

Table 2. Clinical criteria for diagnosing anaphylaxis in adults

Anaphylaxis is likely when any one of the three criteria are fulfilled:	
(1) Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalized hives, pruritus or flushing, swollen lips/tongue/uvula)	
AND AT LEAST ONE OF THE FOLLOWING	
a. Respiratory compromise (e.g. dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)	
b. Reduced blood pressure or associated symptoms of end-organ dysfunction [e.g. hypotonia (collapse), syncope, incontinence]	
(2) Two or more of the following that occur rapidly after exposure to a likely allergen for that patient (minutes to several hours):	
a. Involvement of the skin-mucosal tissue (e.g. generalized hives, itch-flush, swollen lips-tongue-uvula)	
b. Respiratory compromise (e.g. dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)	
c. Reduced blood pressure or associated symptoms [e.g. hypotonia (collapse), syncope, incontinence]	
d. Persistent gastrointestinal symptoms (e.g. crampy abdominal pain, vomiting)	
(3) Reduced blood pressure after exposure to a known allergen for that patient (minutes to several hours):	
a. Systolic blood pressure of less than 90 mmHg or greater than 30% decrease from that person's baseline	

Reproduced with permission [1].
PEF, peak expiratory flow.

reports have described occupational asthma, rhinitis, and contact dermatitis in seafood and crustacean-processing workers [12]. However, OcAn due to seafood/crustacean has been described only in a few case reports [13,14].

OCCUPATIONAL ANAPHYLAXIS DUE TO HYMENOPTERA

In cross-sectional studies [15,16] carried out in beekeepers and forestry workers, the prevalence of IgE-mediated OcAn due to *Hymenoptera* stings was 2–3.4%. Many case reports of anaphylaxis have also been reported in greenhouse and bumblebee farm workers due to bumblebee stings [17]. Other case reports of OcAn due to wasp stings were reported in Spanish gardeners, construction workers, and car drivers [18].

In a recent retrospective study [19[¶]], re-sting data of 96 bee-venom-allergic patients and 95 vespid-venom-allergic patients living in rural Switzerland were analyzed. The risk factors for a wasp or bee re-sting were outdoor occupation, beekeeping, and habitation near a bee house. The authors observed that venom-allergic outdoor workers with systemic reaction face a risk of a more severe reaction when re-stung.

Two reviews on *Hymenoptera* sting reactions, including anaphylaxis and OcAn, have been published in the past 2 years. Tracy *et al.* [20] discussed the growing interest of OcAn due to bumblebees used for pollination in greenhouse workers. In their review, Bilò *et al.* [21] strongly recommended honeybee venom immunotherapy in beekeepers and their family members with positive diagnostic tests and a history of a moderate-to-severe systemic reaction.

OCCUPATIONAL ANAPHYLAXIS DUE TO INSECTS

Anaphylactic reactions after bites of ticks and tsetse flies and contact with pine processionary caterpillars and chironomid larvae used by aquarists as fish food have been reported during occupational or recreational exposure [22–25].

Interestingly, in 2009, an Australian study [26] showed an association between tick bites and red meat allergy in humans. Recent insights on this association have demonstrated that cases of anaphylaxis due to IgE against a mammalian oligosaccharide epitope, galactose- α -1,3-galactose (α -gal), have been associated with delayed-onset anaphylaxis and urticaria 3–7 h after ingestion of red meat (beef, pork, lamb, goat, horse, and kangaroo) [27[¶]]. Cases of delayed anaphylaxis have been identified in

Australia, France, Germany, Sweden, Spain, Japan, Korea, and the United States. Moreover, investigations have shown associations between IgE to α -gal and tick bites that were the causes of IgE responses to α -gal. The α -gal epitope is present in the gastrointestinal tract of ticks (*Ixodes ricinus*) and this implies host exposure to α -gal during a tick bite [28[¶]]. According to the authors, tick bites may cause sensitization to α -gal, with development of red meat allergy as a secondary phenomenon [28[¶]].

OCCUPATIONAL ANAPHYLAXIS DUE TO MAMMALS AND SNAKES

Animal allergens are frequently described as cause of allergic respiratory symptoms and contact urticaria in exposed workers [29], but less commonly as a cause of anaphylaxis. Several cases of OcAn following rodents' bites have been reported among laboratory animal handlers, technicians, and researchers [30,31]. Furthermore, snakebite is acknowledged as an occupational health hazard for snake handlers [32]. In 2012, a prospective observational study [33] was conducted to investigate the epidemiology and clinical presentation of snakebites and envenoming in snake handlers, including workers in a zoo or reptile park, people working with snakes, or undertaking research on snakes, with particular reference to systemic hypersensitivity reactions. Data were obtained by the Australian Snakebite Project that prospectively recruited snakebite patients from over 100 Australian hospitals from 2004 to 2011 [34]. From 1089 snakebites recruited, there were 106 (9.7%) bites in snake handlers and 77 of the 106 patients were envenomed. Eight patients (10%) had an allergic reaction to snake venom and five had reactions satisfying the clinical definition for anaphylaxis, which was severe in three cases. Interestingly, anaphylaxis to antivenom occurred in 7/60 (12%) of treated snake handlers.

OCCUPATIONAL ANAPHYLAXIS DUE TO NATURAL RUBBER LATEX

Following the first report of NRL anaphylactic reaction [35], the number of cases reported rose markedly because of the HIV emergence in the late-1980s. HCW are the most commonly affected, but others include workers of manufacturing plants using NRL and those using NRL gloves in different jobs (e.g. hairdressers, cleaners, and food handlers). In countries and hospitals, the latex epidemic has been partly reduced by primary prevention strategies using latex-free or low NRL protein gloves [36,37].

In 2013, the results of a cross-sectional study [38[¶]] conducted in a rural hospital in South Africa to

determine the prevalence of NRL allergy and the disease spectrum of workers exposed to latex gloves showed a high prevalence of NRL allergy (8.3%); 1 out of 12 workers experienced anaphylaxis requiring mechanical ventilation for 2 weeks.

In 2012, the case of a 51-year-old nurse using NRL gloves has been reported. She referred a 5-year skin manifestation on both hands but never consulted a dermatologist, and during a gynecological examination by a physician using NRL gloves, she suffered anaphylactic reaction [39].

In the same year, an anaphylactic reaction after eating raw manioc, also known as Yuca (an edible root belonging to the *Surge* family of plants), was described in a 46-year-old HCW, with a previous history of severe NRL allergy related to occupational exposure [40]. The authors concluded that manioc allergy was a consequence of primary NRL sensitization. The responsible allergen for this cross-reactivity with manioc was Hev b 5, one of the major NRL allergens whose prevalence is high in HCW [6].

OCCUPATIONAL ANAPHYLAXIS DUE TO CHEMICALS

Chemicals may induce systemic symptoms and/or anaphylaxis in exposed workers, often through unknown mechanisms. Disinfectants, metals, dyes, and bleaches are some of the LMW agents acknowledged as occupational sensitizers causing OcAn [41–43].

A case of OcAn to chlorhexidine was described in 2013 by Wittczak *et al.* [44]. After 22 years of work in the cardiology ward, a 45-year-old nurse developed mild dyspnea and urticaria that appeared periodically during work. Symptoms were ignored until the use of a chlorhexidine-containing toothpaste elicited diffuse urticaria and dyspnea, and then the patient contacted a physician. Laboratory tests revealed a positive skin test to 0.01% solution of chlorhexidine and serum-specific IgE for chlorhexidine; the patient was then submitted to chlorhexidine exposure test. Five minutes of exposure elicited sore throat, hoarseness, throat tightness, mild dyspnea and anxiety, diffuse urticaria over the face and neckline, elevated blood pressure, and tachycardia. Mild epiglottal edema was revealed by direct laryngoscopy. An anaphylactic reaction to chlorhexidine was diagnosed.

MANAGEMENT AND PREVENTION OF OCCUPATIONAL ANAPHYLAXIS

The immediate management of an anaphylactic reaction at the workplace should follow guidelines on anaphylaxis [45,46]. Emergency services should be immediately called when adrenaline is not available, that is, it may especially occur in events

involving workers without any previous anaphylactic reaction. Meanwhile, emergency measures to maintain the airway and provide cardiopulmonary resuscitation should be delivered. Following emergency management, the patients should undergo the diagnostic pathway to identify the culprit agent. Moreover, the patient should be advised to carry two preloaded adrenaline delivery systems with appropriate instructions for use and a MedicAlert bracelet stating 'anaphylaxis, carries EpiPen' (or equivalent) [45,46].

After a diagnosis of OcAn, the patient should be educated to avoid exposure to the culprit agent, both in and out of the workplace. At the workplace, every measure to prevent further exposure should be carried out. A written emergency management plan, health and safety education and training, and surveillance should be enforced in occupations at greater risk.

Exposure elimination, including removal from exposure to the dangerous agent and relocation of the workers, is the mainstay of prevention [47]. However, if this action is not completely achievable, the risk should be minimized, for example, avoiding the direct manipulation of the culprit allergen or wearing protecting gloves or clothings. The treatment of a concomitant contact dermatitis is also advisable as it restores the integrity of the skin surface [47]. In a worker with allergy to *Hymenoptera* venom and a history of venom-triggered anaphylaxis, immunotherapy for *Hymenoptera* venom should be considered, in combination with therapeutic emergency kit [48] because, as also shown by a recent systematic review, it may reduce the incidence of systemic reactions [49].

Anaphylaxis due to nonoccupational allergens, occurring at or exacerbated by the workplace, can be common. In a European survey, 5.9% of food anaphylaxis occurred at workplace [50]. Therefore, in any occupational setting where there is a worker with a history of previous anaphylaxis, the accessibility to an emergency written action plan as well as the availability of adrenaline and a person trained in its administration are mandatory [46].

CONCLUSION

OcAn is a serious event that may be fatal, but its prevalence is not well known. Several HMW and LMW agents have been reported as cause of OcAn in various occupations, but some of them are at greater risk (e.g. beekeepers and HCW handling NRL compounds). In any workplace where there is a worker with a diagnosis of OcAn, prevention of further episodes through elimination or reduction of exposure should be performed. In beekeepers, immunotherapy should be considered. In every

occupation, and, especially, in those at greater risk, a written emergency management plan of anaphylaxis episodes, along with the availability of adrenaline, and trained personnel are mandatory.

Acknowledgements

None.

Conflicts of interest

There are no conflicts of interest.

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