WORK-RELATED ANAPHYLAXIS

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ABSTRACT

A definition of anaphylaxis was recently agreed to at an international symposium on this subject. This article proposes a definition for work-related anaphylaxis that is conceptually consistent with similar classifications for work-related asthma and rhinitis, which defines two major categories – occupational anaphylaxis and work-exacerbated anaphylaxis. The epidemiology and causative agents implicated in work-related anaphylaxis are outlined, with a focus on the most commonly implicated agents such as natural rubber latex, insect venoms, food proteins, disinfectants and pharmaceutical drugs. Diagnosis, management and prevention are discussed. Prevention of work-related anaphylaxis revolves around making a concerted effort to identify the trigger so that more effective primary, secondary and tertiary interventions can be implemented.

DEFINITION

Anaphylaxis is a severe, potentially fatal, systemic allergic reaction that occurs suddenly after contact with an allergy-causing substance.[1] Anaphylaxis and acute allergic episodes manifest clinically with a spectrum of symptoms and signs. This diagnosis has historically been made on a subjective basis with no universally agreed definition or clinical criteria. Recently, a definition of what constitutes anaphylaxis as opposed to other types of allergic reaction was agreed upon at a symposium on the definition and management of anaphylaxis.[2] The symposium proposed the following broad definition useful to both the medical and lay community: **'Anaphylaxis is a serious allergic reaction that is rapid in onset and may cause death'**. The clinical criteria in fulfilling this definition are outlined in Table I. Having precise clinical criteria for the diagnosis of anaphylaxis now makes it possible to conduct multicentre trials and evaluate clinical and epidemiological data more accurately. This in turn will also allow for a more accurate understanding of the role that occupational exposures play in anaphylaxis. Finally, this better understanding may allow improved clinical management and workplace control of these exposures, leading to the prevention of serious anaphylactic reactions in at-risk working populations.

There is no universally agreed upon definition of **work-related anaphylaxis.** However, this entity could be classified into two main categories based on the direct causal relationship between work exposure and the development of the disease: (i) occupational anaphylaxis; and (ii) work-exacerbated anaphylaxis. The generally accepted definition of work-related asthma is categorised similarly.[3] *Occupational anaphylaxis* could be defined as 'anaphylaxis arising out of causes and conditions attributable to a particular work environment and not to stimuli encountered outside the workplace'. Work-exacerbated anaphylaxis could be defined as pre-existing or concurrent allergy (e.g. food/pollen allergy) to a particular agent that is precipitated by workplace exposures, possibly as a result of cross-reacting allergens. In occupational anaphylaxis the exposure could be due to a known or unknown allergen as a result of inhalation, dermal contact (in a person with pre-existing skin disease, e.g. dermatitis, skin trauma) or through hand-to-mouth ingestion in workplaces with poor industrial hygiene practices.

Anaphylaxis usually occurs within 20 minutes of exposure to the causative substance, although occasionally with orally ingested substances, there can be a latency of up to 2 hours between exposure and response.[1,4] If anaphylaxis occurs in the workplace, it is therefore highly likely to have been due to a workplace exposure. In contrast, ingestion-related allergic conditions due to

Table I. Clinical criteria for diagnosing anaphylaxis in adults

Anaphylaxis is highly likely when ANY ONE of the following three criteria is present:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalised hives, pruritus or flushing, swollen lips-tongue-uvula) and at least one of the following:
   a. Respiratory compromise (e.g. dyspnoea, wheeze-bronchospasm, stridor, reduced PEF, hypoxaemia)
   b. Reduced BP 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. generalised hives, itch-flush, swollen lips-tongue-uvula)
   b. Respiratory compromise (e.g. dyspnoea, wheeze-bronchospasm, stridor, reduced PEF, hypoxaemia)
   c. Reduced BP or associated symptoms (e.g. hypotonia (collapse), syncope, incontinence)
   d. Persistent gastrointestinal symptoms (e.g. crampy abdominal pain, vomiting).
3. Reduced BP after exposure to known allergen for that patient (minutes to several hours):
   systolic BP of less than 90 mmHg or greater than 30% decrease from that person's baseline.

PEF- peak expiratory flow; BP – blood pressure.

Adapted from Sampson *et al*.[2]

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exposures outside the workplace may manifest while at work, and therefore may require additional evidence before being labelled 'occupational' or 'work-aggravated'. It also needs to be borne in mind that workers may primarily be sensitised from workplace-allergen exposure, and only manifest with anaphylactic reactions in non-workplace contexts. This is the case with health-care workers undergoing surgical/dental procedures or workers ingesting food or medication after they have developed initial workplace sensitisation and minor occupational allergic symptoms (e.g. rhinitis, urticaria, mild asthma).

EPIDEMIOLOGY AND CAUSATIVE AGENTS

The epidemiology of occupational anaphylaxis is difficult to describe, as the condition is uncommon, transient and previously poorly defined.[5] Globally, it is estimated there are about 154 fatal episodes of anaphylaxis per 1 000 000 hospitalised subjects.[6] Based on data from Olmsted County in the USA, it is projected that there are 84 000 anaphylaxis cases and 840 fatalities in the USA annually.[1,7] Of the total fatalities, it is estimated that about 20% are food-induced (mainly nuts), more than 50% are due to β-lactam antibiotics and less than 10% are from insect stings. There are no reliable figures for South Africa. If the American figures are stratified to adults only, and antibiotics and most food-induced reactions are excluded, it is probable that less than 20% of all anaphylactic fatalities in the USA are due to work-related substances.

Any workplace agent capable of causing occupational asthma or generalised urticaria could theoretically cause anaphylaxis.[8] There are a few clinical case series or reports and epidemiological studies that have been reported in the literature, including those related to fatal occupational asthma.[9] These are outlined in Table II.

One of the most common workplace agents reported to give rise to occupational anaphylaxis is natural rubber latex exposure, especially among health-care workers and latex-manufacturing plant workers where it is used in the production process (Fig. 1). Sensitisation to natural rubber latex in the general population ranges between 5% and 10% with the prevalence in health-care workers varying from 0.5% to 17% on either skin-prick testing (SPT) or latex specific IgE immunoassay.[10] Cumulative incidence rates for latex-induced sensitisation from various studies have been reported to be less than 2% per year with incidence rates of latex allergy being far less, in the order of 1-12 per 10 000 workers per year.[10] There are no reliable figures for rates of *occupational latex-induced* anaphylaxis found in the literature, although a recent study of hospital workers at an academic hospital recorded that 3% of respondents (*N* = 277) reported having anaphylactic reactions.[11] There are however a number of case reports and case series of fatal anaphylaxis due to latex-containing products among health-care workers undergoing dental or surgical procedures or wearing gloves over disrupted irritated eczematous skin.[12]

Table II. Causative agents implicated in occupational anaphylaxis

Agent	Industry
Natural rubber latex (NRL)	Health care Other manufacturing plants with NRL in the production process
Insect (e.g. bees, wasps) and arachnid (e.g. ticks) venom	Honey (beekeepers) Agriculture, parks and forestry, gardening and landscaping
Food proteins (e.g. nuts, seafood, spices, cereal grains, soybean, cow's milk powder and hen's egg powder)	Food-processing industry
Pharmaceutical agents (e.g. β-lactam antibiotics, cytotoxics, laxatives)	Pharmaceutical manufacturing plants Health-care institutions (preparation of medication)
Disinfectants (e.g. chlorhexidine, ortho-phthalaldehyde – OPA)	Health-care institutions Other industries using disinfectants
HBTU (o-(benzotriazol-1-yl) -N,N,N',N'-tetramethyluronium hexafluorophosphate)	Peptide synthesis plants

Fig. 1. Natural rubber latex exposure during surgical procedures in theatre.

Agricultural workers and other outdoor workers are at increased risk of insect stings and venom-induced anaphylaxis (Figs 2 & 3). A Spanish case series of 98 patients with anaphylaxis due to wasp stings reported that 18% of these reactions occurred during working hours.[13] A number of studies on beekeepers have also reported increased rates of sensitisation and allergic reactions to hymenoptera venom, with a prospective cohort study in Greece suggesting a threefold increased risk of sensitisation in beekeepers as compared with non-exposed workers,[14] and a Finnish study reporting approximately 30% of a population of 102 beekeepers having had a previous 'systemic' reaction.[15] Tick-bite-induced anaphylaxis due to *Rhiphicephalus* sp. in a goat herder has also been reported.[16]

Fig. 2. Hymenoptera – honey bee.

Food-related anaphylaxis is a potential problem among workers in the food-processing industry. Food-related anaphylaxis in the domestic environment is commonly due to peanut or other tree-nut allergies.[1] Occupational anaphylaxis in the workplace environment is commonly triggered by inhalation of allergenic food proteins, enzymes (e.g. papain), additives (e.g. sulphites) and food colourants (e.g. carmine) in dust particulate (powder, granules) generated during food-processing (e.g. milling, blending) activities. Severe allergic reactions to a range of inhaled allergens from fish, shellfish, soybeans, seeds, beans and cereal grains, as well as cow's milk and hen's egg powder have been reported in the literature.[17] Spices such as garlic[18] and coriander[19] have also been reported to cause anaphylaxis and could be a potential risk in

Fig. 3. Exposure to bees during beekeeping activities.

Fig. 4. Garlic dust exposure during milling, blending and packing procedures in a spice mill.

workers involved with milling, mixing and packaging spices in food-processing plants (Fig. 4).

Pharmaceutical agents are an important cause of anaphylaxis in the general population, but would also be of concern to workers in pharmaceutical plants (e.g. milling, granulation) and health-care workers involved in the preparation of medication for patient administration. There have been reports of severe allergic reactions to β-lactam antibiotics (e.g. penicillins, cephalosporins), antineoplastic agents (e.g. Adriblastina – doxorubicin hydrochloride) and laxatives (e.g. psyllium) where there is the potential for inhalation of powdered dust particulate or hand-to-mouth ingestion.[20,21]

Finally, there have been isolated case reports of occupational anaphylaxis to a variety of other substances found in the workplace, including chemicals such as disinfectants (e.g. chlorhexidine, ortho-phthalaldehyde – OPA) used in health care settings and HBTU (o-(benzotriazol-1-yl)-N,N,N',N'-tetramethyluronium hexafluorophosphate), which is extensively used for solid and solution-phase peptide synthesis.[22-24]

DIAGNOSIS AND IDENTIFICATION OF TRIGGERS

The diagnosis of occupational anaphylaxis is made according to the criteria set out for the diagnosis of anaphylaxis[2] in the context of an exposure to a suspected workplace agent (Table I). A concerted effort should be made to identify the causative agent because this has major implications for placement of the worker since the removal and relocation of the affected individual from the putative exposure is of prime importance to prevent recurrence. The process to confirm the diagnosis as suggested by Simons *et al.* [25] should be followed: (i) confirm the diagnosis; and (ii) confirm the anaphylaxis trigger.

Confirm the diagnosis

In confirming the diagnosis it is important to work through the following steps:

- Retake a history of the episode, focusing on the antecedent clinical symptoms and signs and obtaining collateral information from fellow workers.
- Review the relevant medical records from the ambulance, emergency department, occupational health clinic, etc.
- Review the laboratory tests (e.g. serum total tryptase, plasma histamine) performed during the episode.
- Review the differential diagnosis, which commonly includes hives, asthma, anxiety/panic attack, fainting, choking.

Identify the anaphylaxis trigger

With regard to confirming the anaphylaxis trigger, there are a number of issues to consider after the episode when the worker has recovered from the acute phase:

- Retake a history of the episode and pay particular attention to questions about inhalational exposures in the 30 minutes prior to the episode and potential ingestion-related exposure within 2 hours. Think about the production process and job tasks the worker performs and create a list of potential exposure agents. In addition review the Material Safety Data Sheets (MSDS) of products the worker may have used.
- Retake a complete medical history, looking for concomitant diagnoses such as asthma, cardiovascular disease, and concurrent medications such as β-blockers, angiotensin-converting enzyme (ACE) inhibitors and others.
- Perform skin tests – skin-prick tests for foods and other agents (e.g latex) and intradermal tests for β-lactam antibiotics. It is preferable for these tests to be done under controlled conditions.
- Perform allergen specific IgE quantitative measurements (Phadia ImmunoCAP Specific IgE) (e.g. insect venoms, cereal flours, spices) and cellular antigen stimulation test (CAST) where appropriate. Identification of cross-reactive allergens may be necessary as well (for instance, latex cross-reactive allergens such as banana, kiwi, pear and avocado; pollen cross-reactive allergens with spices).
- Challenge tests may be indicated that are either allergen specific (e.g. inhalation challenge tests with food products, medication – proceed with extreme caution) or allergen non-specific (e.g. cold and exercise).
- Other assessments as indicated, such as industrial hygiene measurements in the workplace.

It is important to note that SPT and specific challenge tests may precipitate an anaphylactic reaction in sensitised workers and should only be conducted in expert hands if indicated. Specific IgG immunoassays may be a safer, useful alternative if the intention is to rule out exposure to a particular allergen. However, the presence of allergen-specific IgG does not indicate the presence of an allergic cause. The specificity and sensitivity of each allergological test and its correlation with health effects varies between tests. Discussion with the allergologist and laboratory technologist can help the practitioner decide which tests are the most appropriate, taking the clinical context and potential workplace exposures into account. After proper investigation, it would be rare for practitioners to be left labelling the reaction as 'idiopathic' occupational anaphylaxis.

MANAGEMENT

The initial immediate management of an occupational anaphylactic reaction is no different to a non-work-related anaphylactic episode. A recent statement by the World Allergy Organisation concluded that self-administered intramuscular adrenaline is still the mainstay of treatment for anaphylaxis, although it is underutilised and often suboptimally dosed to treat anaphylaxis.[26] Intramuscular adrenaline injection into the lateral thigh is the treatment of choice and it is preferred to intravenous or subcutaneous injection.[27,28] A recent Cochrane review reported that there is no good evidence as to the benefit of antihistamines in the initial treatment anaphylaxis.[29] A more in-depth review of treatment issues of anaphylaxis is dealt with elsewhere in this issue.

Follow-up management of the anaphylactic episode requires relocation and placement of the worker in an area of no exposure after determination of the causative agent in the workplace, so as to prevent repeated exposure of the affected worker. Vigilance regarding other, as yet unaffected, workers is necessary.

Finally, all cases of occupational anaphylaxis must be initially reported to the Compensation Commissioner, Department of Labour, as an occupational disease. The relevant Compensation of Occupational Injuries and Illnesses Act (COIDA) forms should be completed by the medical practitioner and the employer, and the case followed up until finalisation of the compensation process and as the clinical situation dictates. There may be discussion over whether the incident is classified as an occupational injury (a once-off event due to a single exposure) or an occupational disease. If this is the case, the claim is initially managed as an occupational injury and subsequently evaluated as an occupational disease claim should the disease progress to a known compensable entity, such as occupational asthma. Details of this Circular Instruction 176 have previously been published in *Current Allergy & Clinical Immunology*.[30]

PREVENTION

Prevention in relation to the natural history and prognosis of occupational allergy forms a cornerstone of dealing with occupational anaphylaxis. The longer the exposure and delay in diagnosis and treatment, the longer the duration of allergic symptoms, which is ultimately associated with a poorer prognosis and an increased risk of an anaphylactic episode on re-exposure to the offending agent. Risk factors to be considered for modification include environmental factors (exposure to causative or sensitising agents) or host-related factors (atopy, pre-existing food allergies, prior episodes of anaphylaxis, severe uncontrolled asthma, cardiovascular disease).

Primary prevention focuses on prevention of primary or repeated exposure to sensitisers resulting in sensitisation, whether at the source (elimination, substitution, local exhaust ventilation), along the path (enclosure of emission source) or at the worker level (administrative controls, respiratory protective equipment). While respirators may reduce exposure, they are not effective in preventing exposure. All efforts must be aimed at utilising the expertise of experts with insight into the production process, such as engineers and occupational hygienists, to find alternative ways to substitute or eliminate the agent, or reduce exposures to the agent/s concerned. For instance, with natural rubber latex this would entail making the environment latex-free. In some production processes this is not always possible, but attempts to reduce airborne concentrations of the causative agent should always be made. With latex this may involve changing from powdered latex gloves to powder-free low-protein latex gloves to reduce airborne latex particles. While threshold limit values for certain workplace allergens (e.g. latex, flour dust, isocyanates) exist, even low-level exposures have the potential of triggering an allergic reaction in a sensitised worker. In a food-processing worker with a known allergy to the food product, avoidance of the offending food allergen in the diet is another consideration. Similarly, the use of latex-free surgical or dental procedures is indicated in a health-care worker with a known allergy to latex.

Secondary prevention focuses on the prevention of clinical allergy and anaphylaxis in sensitised but asymptomatic individuals. This is effected through early detection of sensitisation to workplace allergens and the presence and degree of impairment of target organs by medical surveillance of workers using questionnaires, SPT, serum-specific IgE, spirometry and other relevant tests to predict future anaphylaxis.

Tertiary prevention focuses on optimal management of a worker with work-related allergy and anaphylaxis to prevent further recurrences and disability. The aim is to reduce the risk of death or reduce the severity of an anaphylactic attack by issuing the worker with an EpiPen for self-administered intramuscular adrenaline injection and a Medic Alert bracelet, and ensuring fellow workers are trained in first aid procedures. Other strategies include removal from ongoing exposure, avoiding exposure to cross-reactive allergens and consumption of food containing the offending allergen or additive, medical monitoring, optimising allergy and asthma treatment, and immunotherapy where appropriate. For bee and wasp venom allergies, desensitisation by means of immunotherapy to hymenoptera venoms has been used with success.[31]

Declaration of conflict of interest

The authors declare no conflict of interest.

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