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REVIEW PAPER

OCCUPATIONAL ANAPHYLAXIS TO FOOD

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Abstract

Increasing incidence and importance of allergies in everyday life leads to bigger impact on the professional lives of patients. Continuous exposure to various allergens may be a trigger factor of development allergic diseases, even anaphylactic shock. This paper summarizes available knowledge of occupational anaphylaxis to food. This narrative literature review was based on selected, peer-reviewed research papers, review articles and case reports between 1997–2024. The aim of the review article was to present the growing problem of allergy patients working in the food industry and to draw attention to the danger of anaphylaxis. In addition to the most popular food allergens, such as milk protein, fish, crustaceans and eggs, authors presented less popular, but becoming more important, such as crickets. Due to the increasing popularity of alternative food sources, the significance of food allergies will become more important. It is worth mentioning that occupational anaphylaxis is not only limited to the exposure to the allergen in the workplace. It can happen outside the workplace as a result of repeated exposure, after re-exposure. Med Pr Work Health Saf. 2024;75(6):521–530

Key words: occupational disease, hypersensitivity reaction, occupational anaphylaxis, anaphylaxis to food, immediate allergy, work-related agents

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INTRODUCTION

The incidence of allergic diseases has increased over the last 30 years and remains one of the most serious public health challenges. This problem has also implications for workers in a wide range of industries and sectors [1]. Exposure to various allergens in the workplace can be a trigger factor in the development of allergic diseases. Food processing appears to be the most common cause of contact urticaria and work-related asthma. The incidence of occupational asthma and contact dermatitis in seafood workers ranges 7–36% and 3–11%, respectively [2]. The majority of occupational anaphylactic relations were described as case reports.

Anaphylaxis is the most severe form of allergic reaction. According to the World Allergy Organization definition, anaphylaxis is the most severe hypersensitivity reaction (grade 3–5) that usually has an acute onset, persists ≥20 min and can lead to death [3,4]. Anaphylactic reactions affect ≥2 systems, usually skin, respiratory tract, circulatory system and/or gastrointestinal tract [2] and may cause severe airway obstruction and/or circulatory insufficiency [3]. There is a lot of data showing a clear link between occupation and anaphylaxis through skin contact and/or airways exposure [5]. Occupational anaphylaxis has a clear socio-economic impact – affected workers may be forced to change their job, their workplace or the type of work. Employees affected by occupational allergies are more likely to take sick leave which not only affects their work efficiency, but can also lead to the need to find new employees due to frequent absence [1].

It is important to note that occupational anaphylaxis is not just limited to the exposure to the allergen in the workplace. It can happen outside the workplace as a result of repeated exposure, after re-exposure [6]. The vast majority of current knowledge on anaphylaxis epidemiology is contained in the anaphylaxis registry, which has been established in Germany since 2017 and includes altogether 5851 cases [7]. Among them only 225 (3,8%) were assigned to be caused by an occupational allergen. The majority were insects (82,79%), food (12%) and drugs (3,6%). Therefore, all reactions need to be carefully assessed for occupational triggers.

METHODS

This narrative literature review was based on selected, peer-reviewed research papers, review articles and case re-

ports between 1997–2024. After careful searches for "occupational anaphylaxis," "food anaphylaxis" and analysis of papers published in PubMed, Medline and Cochrane search, only 64 that met the criteria. Authors focused on papers published in English, Polish and German. There are few publications describing a given sample, but authors hope to expand the research to include other food products.

RESULTS

Pathogenesis

A food allergy is defined as an adverse immune reaction to food. It includes immediate IgE-mediated (immunoglobulin E) hypersensitivity reactions, delayed IgE-independent reactions, and disorders involving both pathways. They cause symptoms, such as urticaria, wheezing, cough, shortness of breath, weakness, nausea, vomiting and diarrhea. Anaphylaxis refers to a severe allergic reaction that affects ≥2 systems [8].

The onset of allergy occurs in early childhood. Food allergy begins frequently in early childhood (according to various sources, the incidence of allergies in children is 6–8%), which may later progress to atopic dermatitis (AD), bronchial asthma or allergic rhinitis (the so-called allergic march) [7]. This is the natural history of the development of atopic diseases according to the criteria proposed by Wickman [7]. At the same time, it should be remembered that there are also other forms of the so-called allergic march. The order of atopic diseases in a different allergic march may be reversed, the order in which allergic conditions occur may be completely random or begin with a disease other than food allergy or AD [7]. The incidence of atopy is influenced not only by genetic factors, age at onset, but also by proper skin care in this period [9]. Even infants with temporary skin barrier disorders, skin microbiome dysbiosis and/or persistent AD are particularly exposed to the risk of developing food allergy (during the allergic march) [9].

Numerous studies have shown a relationship between AD and allergies to food allergens. Epidemiological data confirm the progression of the allergic march from AD to food allergies [10–13]. It has been described that a damaged epidermal barrier and AD precede the occurrence of food allergy [14–17].

Genetic predisposition leads to epidermal barrier dysfunction and subsequent inflammatory damage with the involvement of Th2 (Type 2 helper 2 cells) lymphocytes causing the release of pro-inflammatory cytokines which promote water loss, persistence of inflammation and allow allergen to penetrate into the deep layers of the skin. Local dendritic cells (Langerhans cells) which are antigen presenting cells capture food allergens that penetrate skin-layers [14–16].

The mechanisms underlying anaphylaxis (both occupational and non-workplace) may be allergic, IgE-dependent, IgE-independent or non-allergic [6]. High molecular weight allergens, such as cereal, crustacean, fish, flour and spice allergens, typically induce IgE-mediated reactions. Some low molecular weight allergens, such as epigallocatechin gallate found in green tea, can also cause IgE mediated anaphylaxis. They can also act as a hapten as they are able to penetrate the skin and bind to the proteins in the epidermis [13].

Depending on the route of exposure, symptoms of anaphylaxis usually appear within a short time. The possible influence of cofactors, such as physical exertion, cold, stress, medication, concomitant infection, should be considered. Workers who are allergic to occupational allergens may develop anaphylaxis outside the workplace as a result of exposure to the same or cross-reactive allergens. If identification of the allergen is problematic, cofactors should be considered. Special attention should be paid to exercise-induced food anaphylaxis, especially in manual workers [13].

Workers allergic to occupational allergens may also develop anaphylaxis outside of the work environment as a result of exposure to the same or cross-reactive allergens. If it is difficult to identify the allergens that caused anaphylaxis, cofactors, such as infection or the use of nonsteroidal anti-inflammatory drugs, should be taken into consideration, as they may increase the risk of its occurrence (summed anaphylaxis).

Allergens

Fish and crustacean

The increasing number of patients attend an allergist with symptoms of hypersensitivity to seafood is most likely related to the constantly increasing consumption of seafood around the world [18]. Regardless of this phenomenon, allergies are observed among workers in the fishing industry, who are exposed to frequent contact with fish and seafood allergens. Allergic reactions in various forms are observed in workers in the fishing industry who have a stable exposure to seafood allergens. Exposure is mainly via the skin or the respiratory tract by inhalation of vapours or aerosols when preparing meals or cleaning rooms where seafood is stored and/or processed [2].

Seitz et al. [19] described the case of a lorry driver who transported seafood, but repeatedly ignored using

personal protective equipment (protective clothing and gloves) because of time pressure. This led to increased direct contact of the skin and mucous membranes with allergens. As a result of the gradual worsening of his skin symptoms (contact urticaria), his employer decided to transfer him to another job. From then, he had to wash fish transport tanks with pressurized water. On his first day of work, he suffered a severe bronchospasm, laryngeal oedema and anaphylaxis so he requested emergency treatment. This case report clearly shows that regular exposure to a specific allergen in the workplace can gradually lead to more severe forms of allergic reaction [19].

Another problem among employees of the fishing industry is allergy to *Anisakis simplex* (a species of large nematode parasitizing fish, including herring, zander, cod and salmon). The available literature describes cases of fish sellers and cooks who developed symptoms of hypersensitivity after contact with infected food [20–23]. The patients consulted a doctor because of itching and hives, occurring mainly on the upper limbs. In these patients, fish allergy is often suspected, but skin prick tests and specific IgE are negative. In this case it is useful to perform diagnostic tests for *Anisakis* allergy [22].

Other species of fish parasites, such as *Hysterothylacium*, *Capillaria gracilis* and *Cucullanus*, have been proven to cause cross-reactions with *Anisakis simplex*. There are data suggesting that in ≤50% of patients this cross reactivity is clinically relevant [21,22]. In patients with symptoms of anaphylaxis to *Anisakis simplex*, complete cessation of consumption fish and crustaceans is recommended [23].

Buckwheat

Buckwheat is a popular allergen especially in Eastern Asia countries (Korea and Japan), but its importance is increasing as it is allowed to be included in gluten-free diet. The first reports of occupational allergy to buckwheat in production of pasta and pancakes were described in Japan, Korea and some European countries [24–26]. Occupational inhalant allergy to buckwheat has been the subject of some epidemiological studies [27]. One of these, conducted in Sweden, showed a high incidence, approx. 28% of diagnosed buckwheat allergy in workers who inhaled buckwheat dust during the repackaging of buckwheat and buckwheat flour. The total exposure to dust suspended in the air during the repacking of buckwheat was within the applicable exposure standards for organic dust (1.7 mg/m^3 with the standard <5 mg/m^3). These patients benefit greatly from the use of protective masks [25]. Buckwheat allergy is a common cause of anaphylaxis [28–30]. Occupational exposure occurs mainly in bakeries and other food processing industries. Jungewelter et al. [29] described series of cases of bakers, cooks and grocery store workers who had prolonged exposure to buckwheat flour. The workers used rubber gloves but none of them used respiratory protection. Jungewelter et al. [29] described series of 6 cases of bakers, cooks and grocery store workers who had prolonged exposure to buckwheat flour and developed anaphylaxis at work. The diagnosis was confirmed by skin prick tests and elevated serum specific IgE antibodies. Three of the 6 patients living in Denmark, suffered from anaphylaxis after eating pancakes, blinis and bread containing buckwheat [31]. It is worth paying attention to the fact that 1 of the bakers suffered from 2 episodes of anaphylactic shock with a 2-day interval between them. The first episode occurred after eating blinis, and the next after exposure to buckwheat flour at work. Serum concentration of specific IgE to buckwheat in this patient was >100 kU/l. It is worth noting that all the patients who developed anaphylaxis, previously suffered from rhinitis, coughing and dyspnoea. The authors conclude that the high rate of severe allergic reactions and the short exposure time to sensitization are unique features of buckwheat allergy [32].

The main allergens in buckwheat (*Fagopyrum esculentum*) are Fag e 1, Fag e 2, but recent data show that Fag e 3 also has an important role in allergic reactions in susceptible individuals. Component resolved diagnosis is a precise laboratory tool when allergy is suspected in workers. Clinically relevant cross-reactivity has been reported between buckwheat and peanut, latex, coconut, quinoa and poppy seed [33]. Fag e 2 appears to be responsible for cross-reactivity with latex and legumes. Therefore, despite the high risk of anaphylaxis, the provocation test remains the gold standard for the diagnosis of food allergies [33].

Milk

Cow's milk protein allergy in adults is much rarer than in children. In the former, the most common allergen is casein, while in children – lactalbumin [34]. In authors' professional practice, several times they have observed food industry workers who developed acute contact dermatitis while working with powdered milk. Cases of these patients suggest that the sensitization process initially affecting the skin and respiratory tract may lead to a generalized reaction over time.

Prolonged exposure to cows' milk proteins may cause dyspnoea, occupational asthma and systemic reactions

in some individuals over time. Sierra et al. [35] described the case of a 62-year-old patient with 17-year history of working in a diary industry who suffered from anaphylactic shock caused by accidental ingestion of milk. She had worked without protective equipment. Two years after starting work with milk powder she developed contact eczema. The patient completely eliminated cow's milk proteins from her diet. Skin prick tests and positive specific IgE to milk proteins confirmed the association between occupational exposure and anaphylaxis [35].

The case of this patient suggests that the sensitization process initially affecting the skin and respiratory tract developed to a generalized reaction over time. There are very few reports of occupational anaphylaxis to dairy products, but it is important to remember that such exposure may occur in the workplace.

Egg

There are not many reports on occupational hen egg allergy. One of these reports describes the history of a 54-year-old poultry industry worker [36]. He had been regularly exposed to hen and quail feathers, faeces and eggs for 17 years. Approximately 10 years after starting work the patient developed cough, dyspnoea and wheezing which resolved outside the workplace. A few years later after eating cooked quail egg, he developed generalized urticaria with severe pruritus, oropharyngeal oedema, dyspnoea and stridor. Skin prick tests with hen egg (egg white, yolk, ovalbumin, ovomucoid) and chicken meat, as well as specific IgE assays were negative. An open oral food challenge was performed with hen egg, and it was also negative. The main allergens in quail egg are ovalbumin, ovomucoid and ovotransferin, but these proteins do not cross-react with hen egg [1,36]. Quail egg allergy is described as very rare.

The prevalence of sensitization to egg in adults ranges 0.35–1.9% and is reported mainly in the bakery and confectionery industries, where workers are exposed to egg protein aerosols [36].

Chicory

Two types of chicory are mainly used in the food industry: common chicory, which is used as a coffee substitute, and true endive chicory which is eaten raw. Both species may cause contact dermatitis and/or anaphylaxis [37,38]. Willi et al. [39] reported the case of 35-year-old cook who suffered from chronic hand and forearm eczema, but also experienced several episodes of generalized pruritus, urticaria and shortness of breath. All symptoms improved significantly away from the workplace. Skin prick tests to inhalant and food allergens, and specific IgE to inhalant and food allergens were negative [39]. However, skin tests with food the patient had contact with at work, showed strongly positive reaction to *Cichorium intybus*. Serum total IgE was elevated but specific IgE on a standard food panel was negative. Patch tests with the primary and additional series of allergens gave no reaction, but chicory caused a strong positive reaction with erythema and a few nodules. The patient confirmed repeated contact with chicory, which always preceded the onset of symptoms. Therefore, he was informed to avoid chicory and emergency medications were prescribed (adrenaline, steroid and antihistamine). By following the recommendations, contact dermatitis disappeared quickly and completely. After an asymptomatic period, the patient was exposed to cooking chicory vapour and immediately developed generalized itching, erythema and dyspnoea, which indisputably confirmed IgE-mediated type I allergic reaction to chicory [39].

Spices

Most allergic reactions to spices are the result of cross-reaction with pollen allergens. In particular, mugwort and birch pollens increase a risk of spice allergies [40,41]. Occupational exposure and allergic symptoms occur mainly among butchers, bakers, florists and workers in spice factories. Occupational rhinitis, conjunctivitis, and even asthma, have been described in the case of several spices, such as peppers, coriander, fennel seeds, saffron, anise, garlic, onion, rosemary, leek, sesame, thyme and white pepper. Even though occupational exposure in such patients is common (from spice production to spice processing), several cases of occupational anaphylaxis after contact with this allergen have been described.

Ebo et al. [42] described an interesting case report of 25-year-old man who suffered from anaphylactic reaction with generalized urticaria, conjunctivitis, angioedema of mouth and throat and bronchospasm after eating pita bread with yoghurt dressing. He did not suffer from any classical inhalant or food allergy. However, his professional history revealed that he worked as a spice grinder and packer for 3 years before the anaphylaxis incident. While working, used to suffer from urticaria, angioedema with progressive rhinoconjunctivitis and asthma while handling the coriander and fenugreek dust. Exposition to other spices, such as rosemary, saffron, thyme and pepper, remained well tolerated. The patient was not aware of professional health hazards. Analysis of specific IgE to coriander and fenugreek confirmed allergy to both spices. Considering the patient's overall

clinical symptoms and his professional history, it can be concluded that the spice allergy in his case is an occupational disease. In order to avoid allergic reactions patient was instructed about his allergy and possible progression of his condition in case of potential development of cross-reaction with other spices [42].

Due to the high frequency of cross-reactivity in this group of allergens it is essential to recognize all possible cross-reactions and strictly reduce the possible exposure.

Crickets

In conjunction with the growing popularity of insects in the diet, they are increasingly considered as food and occupational allergens. Occupational allergy to crickets may be a problem among workers in reptile food factories, pet reptile owners, zoo workers and chefs preparing dishes with insects. Additionally, several publications describe cricket cross-reactions with seafood [43–45]. De las Marinas et al. [46] describe cases of anaphylaxis after contact with crickets. The 37-year-old cricket breeder fed reptiles, so he had contact with them every day, but he did not use personal protective equipment. After direct contact with the exoskeleton of crickets, he developed rhinoconjunctivitis, contact dermatitis and shortness of breath. Skin prick tests showed positive results for mites, molds and olive pollen. The patient did not show any symptoms of hypersensitivity after contact with grasshoppers, mealworms and cockroaches. Patient was diagnosed using prick-by-prick with 4 cricket species (native tests) such as: *Gryllus assimilis*, *Gryllus bimaculatus*, *Gryllodes sigillatus* and *Acheta domesticus*. Also laboratory tests revealed increased sIgE (IgE antibodies that specifically recognize and bind the allergens against which they were created) levels for all 4 cricket extracts.

The native tests with the same species of crickets confirmed sensitivity to those insects. It is worth noting, that these are the species most commonly used in food production. Laboratory tests revealed increased sIgE (specific IgE) levels for all 4 cricket extracts. Although this problem may seem to affect a small part of the population, it is important to remember the growing popularity of diets containing insects. The rate of insect allergy will increase over time as the insects become more common in the human diet [43–46].

Red meat

Anemüller et al. [47] in 2018 presented the case of 30-year-old cook with history of recurrent episodes of angioedema, shortness of breath and diarrhea occurring several times a month [47]. These symptoms appeared after eating red meat, then touching fresh meat even in the smallest amounts (e.g. tasting beef broth). In this patient, the symptoms were closely related with the amount of allergen consumed – after eating a grunt, the symptoms were more pronounced and affected several systems, if the allergen dose was small – only mild swelling of the lips occurred. Over time, the symptoms became more pronounced and involved several systems. He recalled a tick bite several years earlier, after which, prolonged erythema of the bitten area persisted. Skin prick tests with beef and gelatin showed positive results, whereas skin prick tests with pork remained negative. The attending physician recommended complete avoidance of foods containing galactose-α-1,3-galactose (α-gal) and reported the disease to the appropriate professional association. As a result, the patient was dismissed from work, which prompted the attending physicians to file a medical complaint in case of suspicion of an occupational disease. In their expert opinion, specialists from the dermatological center in Osnabrück confirmed the diagnosis of delayed type of immediate allergy to red meat. Immunoblot analysis confirmed the diagnosis of delayed allergy to α-gal. Symptoms of the disease are caused by professional work. The patient's illness was recognized as an occupational disease. He was retrained to another job. As a result of retraining, he suffers significant financial losses. The patient still avoids eating red meat and gelatin, and carries an adrenaline injector, glucocorticosteroids and dimethinden. He is currently asymptomatic with 1 exception: after ingesting a cold medicine capsule containing gelatin, delayed angioedema of the lips occurs [47].

Unal et al. [48] published the results of oral immunotherapy in 20 patients with α-gal syndrome. All the treated patients were given 100 g of red meat every day for 6 months and then every other day for the next 6 months. After the first year of immunotherapy it was recommended to eat beef meat 2–3 times a week. All the patients who received immunotherapy developed tolerance to red meat. During 5 years of follow-up, the median α-gal specific IgE serum concentration decreased in 9 patients but all of them consumed red meat without any signs of allergy. This study clearly shows the long-term safety and efficacy of α-gal oral immunotherapy, but due to the small number of participants in the treated group further observation is needed.

Cross-reactivity: latex

Latex was a major occupational allergen in the 1980–1990 period, but safety restrictions and regulations led to the wide-

spread use of nitrile, neoprene, polyvinyl and polyurethane in place of latex, resulting in a significant reduction in reported allergic reactions in the workplace within a short period of time [49]. European data from the Anaphylaxis Registry [50] clearly show that occupational latex anaphylaxis is a rare clinical problem. From 2017 to 2023, 5851 cases of suspected occupational anaphylaxis were described, but only 225 (3.8%) were actually triggered by an occupational allergen. Latex anaphylaxis cases were only 2 of them (0.9%) [50]. In some countries with a lower economic status, where latex substitution is not possible, the overall rate of latex allergy is significantly higher. Boonchai et al. [51] reported that the incidence of latex allergy in Thailand is 13.3% higher than in more developed countries. Latex allergy is a risk factor for latex fruit syndrome, which is a consequence of the similarity of the sequence homology between natural rubber latex and certain plant food allergens. Scarce epidemiological data indicate that this problem may affect 4–88% of latex-allergic patients [52]. The highest incidence of latex fruit syndrome was observed in the Finnish population [53]. Symptoms can range from benign signs of oral allergy syndrome to severe anaphylaxis. Ten latex (*Hevea brasiliensis*) allergens: Hev b 5 (acid structural protein), Hev b 6.01 (prohevein), Hev b 6.02 (hevein), Hev b 6.03 (hevein C), Hev b 7 (patatin-like protein), Hev b 8 (profilin), Hev b 9 (enolase), Hev b 10 (manganese superoxide dismutase – MnSOD), Hev b 11 (chitinase class I), Hev b 12 (non-specific lipid transfer proteins – nsLTP) are responsible for latex fruit reactions.

Gromek et al. [52] analysed medical databases in search of an accurate characterization of latex fruit cross-reactivity. Authors screened 401 articles but decided to analyse only 14 of them in detail because of multiple bias. It was concluded that the most commonly reported plant foods responsible for fruit latex syndrome were: banana, kiwi, avocado and papaya. The precise characterization of the clinical manifestations showed that the most common symptoms involved mucous membranes and skin (urticaria, angioedema, AD, pruritus, erythema). Asthma exacerbations and rhinitis were of minor importance and mainly affected people with underlying atopic disease. Gastrointestinal and anaphylactic reactions were less frequent.

Awareness of the potential variability in symptoms, progression and possible outcomes requires the correct use of grading systems [54]. Many of the grading scales proposed by numerous societies and interest groups are still inconclusive. Hopefully, further observation and research will resolve unanswered questions and doubts. At least 10 allergens of *Hevea* are responsible for sensitization in predisposed individuals. All allergens are different in terms of structure, size and immunogenicity. Data suggest that contact with damaged skin may be the trigger for latex allergy [55]. Repeated exposure to latex is a risk factor for severe reactions. Coexisting food allergy to chestnut, pineapple, banana, mango, avocado, melon is another important risk factor for developing latex allergy due to cross-reactivity [56]. The most frequently exposed group are health sector workers, due to contact with personal protective equipment containing latex [57]. West et al. [58] described a case of a nurse who, while staying in 1 room with balloons, developed airway swelling, shortness of breath, cough, and urticaria on her arms. She was previously diagnosed with allergy to pineapple, shellfish, and latex. Two doses of intramuscular epinephrine were given, as well as dexamethasone, famotidine, IV lactated ringers, and 2 nebulizer treatments with racemic epinephrine. After good improvement in symptoms, she was discharged, but on her way home, she again experienced throat swelling, shortness of breath, and facial tingling. Unfortunately, she required another racemic epinephrine nebulized treatment and IV epinephrine. The patient's vital signs stabilized, and after additional observation, she was discharged after 48 h [58]. In some cases, the symptoms of severe occupational latex allergy may be masked by another disease. Lertvipapath [59] described the case of a young nurse suffering from chronic spontaneous urticaria, which complicated the diagnosis of latex-induced anaphylaxis, as the symptoms of both can coexist and lead to misdiagnosis. Today, most data on latex allergy are case reports of healthcare workers or those with frequent exposure to latex. The vast majority of reported cases involved patients with atopy and/or food allergy [60,61]. This observation suggests that these risk factors are crucial for the development of severe latex allergy symptoms. In order to use preventive methods to reduce latex allergy in the workplace, correct diagnostic procedures are essential. However, please remember that broadly understood cross-reactions may occur more frequently.

Diagnostics

The diagnosis of occupational anaphylaxis requires confirmation of a diagnosis of anaphylaxis and identification of the agent inducing the reaction [7]. The diagnosis is based primarily upon clinical symptoms and signs, as well as a description of the acute episode; including antecedent activities and events occurring within the preceding minutes to hours. A key task is to demon-

strate the temporal relationship between the occurrence of symptoms and the work performance: duration of employment in the current position before the first symptoms appeared, the type of substances presented at the workplace and their association with the appearance of anaphylaxis [6]. It is also necessary to carry out detailed analysis of occupational exposure and selection of potential exposures: allergenic, toxic or irritating factors. It is important to perform the differential diagnosis, such as: congenital angioedema, chronic urticaria, chronic obstructive pulmonary disease or even anxiety disorders [1]. Component-resolved diagnosis may help in the identification of primary sensitizers or cross-reactive allergens [6]. The diagnosis of occupational anaphylaxis is based on the medical history, skin prick testing, assessment of specific IgE serum concentration and in selected cases provocation tests which remains the gold standard. If allergic occupational asthma is suspected a workplace-related inhalation test is recommended [3]. It is a specific test in which the diagnosed person is exposed to controlled concentrations to an agent present in the workplace. The assessment of serum tryptase performed during or immediately after an anaphylaxis episode should be reviewed. A real problem in the search for a trigger are hidden occupational allergens (additives). They are usually not declared on product labels and to make the matters worse, their long-term impact on health is still unknown. These compounds are the result of innovative technologies to improve selected properties of food products [6].

CONCLUSIONS

Repeated and frequent occupational exposure through inhalation or/and skin contact may lead to IgE-mediated allergy symptoms. The increase of symptoms may be gradual and last for a longer time. Skin symptoms, which are often underestimated, may precede the onset of anaphylaxis by several years [7,8]. It is important to know that anaphylaxis can be caused not only by ingestion of the food allergen but also by the contact with the skin, mucous membrane or even by inhalation. The clinical picture of anaphylaxis depends not only by the route, but also by the frequency of exposure. Repeated exposure may lead to allergic reaction or to tolerance [48]. One should also not forget about the cofactors of an anaphylactic reaction, such as physical exercise, cold, alcohol, stress or some medications (nonsteroidal anti-inflammatory drugs). Based on the observation that patients become allergic through the skin and/or respiratory exposure, workers should be advised to use appropriate protection. Workers should be informed of the long-term consequences of non-compliance. In workplaces where there is an increased risk of anaphylactic reactions, a specific action plan for life-threatening situations should be prepared. More data is needed to provide more information on reliably diagnostic methods in the search for potential occupational triggers. Consequence and development are necessary to prevent occupational anaphylaxis.

Author contributions

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