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Review

Food-Dependent Exercise-Induced Anaphylaxis: Current Insights and Future Directions

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SUMMARY

Food-dependent exercise-induced anaphylaxis (FDEIA) represents a rare allergic condition in which anaphylaxis is triggered only by the combination of eating specific foods and subsequent physical activity, often influenced by cofactors such as NSAIDs, alcohol, or infections. The pathophysiology involves increased intestinal permeability, enhanced allergen absorption, and immune dysregulation. Management emphasizes acute treatment and, crucially, prevention through patient education and cofactor control. Preliminary evidence supports the role of biologics, such as omalizumab and dupilumab, and oral immunotherapy in selected patients.

KEYWORDS: food-dependent exercise-induced anaphylaxis, exercise-induced anaphylaxis, anaphylaxis

INTRODUCTION

Anaphylaxis is an acute, potentially life-threatening hypersensitivity reaction characterized by the systemic activation of mast cells and basophils, leading to the release of inflammatory mediators [1]. While typically triggered by a single allergen, more complex forms exist that require multiple concomitant factors. Among these, exercise-induced anaphylaxis represents a unique condition in which physical activity, alone or in combination with specific foods, can elicit an anaphylactic reaction [2].

Specifically, in the food-dependent form (FDEIA), the reaction occurs only if food ingestion is followed by physical exercise [3,4]. Although rare, this clinical entity is increasingly recognized during adolescence and young adulthood. Despite recent advances in understanding FDEIA, many uncertainties remain. The interaction among foods, physical exertion, and cofactors is complex and not yet fully clarified, and there is currently a lack of standardized diagnostic protocols.

PHYSIOLOGICAL MECHANISMS

Physical activity plays a key role in the pathogenesis of FDEIA, acting not only as a trigger but also as a modulator of the allergic response threshold. The type and intensity of exercise required to provoke anaphylaxis also vary widely. Reactions have been observed during all stages of physical activity, from warm-up to recovery [2]. FDEIA typically occurs when the interval between food intake and the start of exercise ranges from 30 to 120 minutes, though cases with much longer intervals, up to 6 hours, have been documented [5].

From a pathophysiological standpoint, one of the primary hypothesized mechanisms is increased intestinal mucosal permeability during exercise. This facilitates the systemic availability of allergens by allowing them to cross the intestinal barrier into the bloodstream, enhancing the likelihood of interaction with allergen-specific immunoglobulin E (IgE) bound to tissue mast cells and basophils. Additionally, exercise induces a redistribution of blood

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flow, reducing visceral perfusion in favor of muscles, skin, and heart, promoting contact with more reactive mast cell phenotypes [2]. Increased plasma osmolarity, which has been correlated with amplified histamine release, further contributes to lowering the degranulation threshold [6].

In wheat-dependent exercise-induced anaphylaxis (WDEIA), physical activity activates intestinal tissue transglutaminase, which binds to dietary peptides, promoting aggregation and enhancing IgE cross-reactivity, thereby facilitating more efficient mast cell degranulation. Interleukin-6, produced in response to muscle contraction, may also upregulate transglutaminase expression, further amplifying allergic sensitization [2].

Among other implicated mediators, adenosine, released during exercise, can bind to the A3 receptor on mast cells and basophils, triggering their activation. Moreover, a reduction in prostaglandin E2 levels, observed in some FDEIA patients, may contribute to lower reactivity thresholds, increasing susceptibility to anaphylaxis [7].

THE MOST COMMON ALLERGENS IN FDEIA

Based on data from the European Anaphylaxis Registry, which includes nearly 3,500 cases of food-induced anaphylaxis from 10 European countries, the most common triggers of pediatric food-induced anaphylaxis are peanut, cow's milk, cashew, and hen's egg, whereas in the adult population wheat and shellfish predominate [8]. In Central Europe, wheat accounted for 15% of all food-induced anaphylaxis, with 83% of these reactions occurring in the presence of exercise as a cofactor [9]. A higher prevalence of wheat allergy has been reported in Asian countries. Studies conducted in China found wheat to be the cause of up to 37% of anaphylactic reactions, with 74% of wheat-induced symptoms triggered by physical exertion [10].

Given the frequent occurrence of exercise-induced anaphylaxis following wheat ingestion, this syndrome has been recognized as a distinct clinical entity and is now referred to as WDEIA with wheat beeing the most studied and best-characterized allergen in the context of FDEIA. Other allergens frequently described include lipid transfer proteins (LTPs) and those derived from shellfish. In some patients, anaphylactic reactions occur following the combined ingestion of multiple foods, suggesting a possible synergistic effect between different allergens in lowering the threshold for mast cell activation [11].

WHEAT ALLERGENS

The storage proteins of wheat, including gliadins and glutenins, represent the main allergens involved in FDEIA. Among the best-characterized allergens, ω-5 gliadin (Tri a 19) is considered the principal trigger of WDEIA, capable of inducing anaphylactic reactions both in the presence of cofactors and, in some cases, in isolation [12,13]. Studies have demonstrated cross-reactivity between ω-5 gliadin and other prolamins, such as rye secalins and barley hordeins, suggesting that these cereals may also provoke symptoms in patients with WDEIA [14]. In addition to ω-5 gliadin, other wheat allergens have been implicated in WDEIA, albeit less frequently. High molecular weight (HMW) glutenins and the α-, β-, and γ-gliadin fractions have been associated with reactions in patients testing negative for ω-5 gliadin. Another relevant allergen is the α-amylase/trypsin inhibitor, characterized by heat resistance and the ability to bind specific IgE, and potentially contributing to the pathogenesis of WDEIA [13]. Furthermore, the wheat lipid transfer protein (Tri a 14), known for its thermal stability and resistance to gastrointestinal digestion, may also induce allergic reactions [15].

LIPID TRANSFER PROTEINS (LTPS)

Lipid transfer proteins, belonging to the prolamin superfamily, are characterized by marked thermal stability and resistance to gastric digestion, making them highly persistent allergens. They are widespread in numerous plant-derived foods and represent one of the leading causes of food allergy in Mediterranean countries [16]. LTPs are present in fruits, vegetables, cereals, nuts, tree and grass pollens, as well as in latex, and are concentrated mainly in the peel or outer layers of foods.

Notably, peach LTP (Pru p3) has emerged as the most prevalent allergen, identified in approximately 77% of LTP-sensitized individuals [17]. In some patients, peeling the fruit may reduce reactivity; however, the presence of other cofactors may lower the threshold of reaction, triggering anaphylaxis even without direct exposure to high allergen concentrations [18]. Epidemiological studies in Italy have shown that LTPs are the leading cause of exercise-induced anaphylaxis [19]. In a recent study by Scala, among 116 subjects with FDEIA, 77 (66.3%) were reactive to LTPs, and the severity of allergic reactions correlated directly with specific IgE levels against LTPs [20].

SHELLFISH ALLERGENS

Tropomyosin is considered a pan-allergen among invertebrates due to its high structural homology across different species. This allergen is ubiquitous in shellfish — such as shrimp, crab, lobster, and squid — as well as in other invertebrates including mollusks, mites, and cockroaches, promoting cross-reactivity phenomena with sequence homology reaching up to 98% [21]. Its strong thermal and digestive stability contributes to its clinical relevance in food allergic reactions, making it the main allergen involved in shellfish-induced anaphylaxis. Epidemiological studies report that tropomyosin is responsible for approximately 80% of anaphylactic reactions following shrimp ingestion. An analysis conducted in Hong Kong on a cohort of 131 patients with confirmed anaphylaxis found that FDEIA represented 42% of cases, with shellfish identified as the second most frequent cause after wheat, with an incidence of 16.7% among the subjects analyzed [22].

α-GAL

The carbohydrate epitope galactose-α-1,3-galactose (α-gal) is present in mammalian tissues and is known for its etiological role in the so-called "α-gal syndrome". This allergic condition arises following the ingestion of red meat (such as beef, pork, or lamb) or internal organs, particularly pork kidneys, leading to delayed-type allergic reactions. Beyond food exposure, anaphylactic reactions can also occur after the administration of medications containing α-gal, such as cetuximab and some animal gelatin-based formulations. Sensitization to this molecule is mainly triggered by tick bites, which introduce the antigen via saliva, inducing an IgE-mediated response. Unlike classical IgE-mediated food allergies, symptoms do not occur immediately after ingestion but typically develop 3-6 hours after meat consumption, due to the delayed release of the antigen into circulation following prolonged meat digestion. The role of cofactors is particularly relevant in α-gal syndrome 23). Fisher et al. analyzed a cohort of 25 patients with allergic reactions linked to pork kidney or red meat ingestion, reporting that in 81% of cases the presence of cofactors was decisive in triggering clinical symptoms [24].

SOY

Soy is another allergen involved in the pathogenesis of FDEIA. The responsible allergens vary according to the form of preparation. In cases of FDEIA related to tofu consumption, the main proteins involved are storage proteins, in particular β-conglycinin (Gly m 5) and glycinin (Gly m 6), which have been identified as markers of severe anaphylactic reactions in patients with soy allergy. Conversely, the ingestion of soy milk followed by exercise has been associated with reactions mediated by Gly m 4, an allergen belonging to the PR-10 protein family (pathogenesis-related proteins), known for its cross-reactivity with Bet v 1, the major birch pollen allergen. These observations suggest the existence of two subtypes of soy-related FDEIA: a storage protein-related form, often associated with concomitant peanut sensitization, and a PR-10-related form, typically observed in individuals with birch pollen allergy. Nevertheless, in many patients with soy-induced FDEIA, the precise allergen responsible for the reaction remains uncertain [25,26].

COFACTORS

In addition to food and exercise, cofactors play a crucial role in modulating the threshold for allergic reactions. Notable cofactors include nonsteroidal anti-inflammatory drugs (NSAIDs), which compromise the intestinal barrier and enhance mast cell degranulation [27]. Proton pump inhibitors can increase the absorption of protein allergens by altering gastric pH [12]. Alcohol and its metabolites disrupt cellular junctions and potentiate mast cell activation [27]. Hormonal factors, such as menstrual cycle variations, can also affect vascular permeability and immune responsiveness. The gut microbiota, particularly through butyrate production, further influences immune tolerance to allergens [28].

Additionally, psychophysical stress, dehydration, viral or bacterial infections, and sleep deprivation may act as indirect cofactors, amplify immune responses and triggering FDEIA even in the presence of allergen doses that are normally tolerated [29,30].

DIAGNOSIS AND THERAPEUTIC STRATEGIES

The diagnosis of FDEIA primarily relies on a detailed clinical history, which is the most effective tool to identify the temporal association between food ingestion, physical exercise, and symptom onset. Gathering specific information about the sequence of events, the nature and quantity of the ingested food, timing of physical activity, and potential cofactors is essential.

The diagnostic process includes skin prick tests with suspected allergens and serum-specific IgE assays. Component-resolved diagnostics, such as ImmunoCAP, support a precision medicine approach in FDEIA and may be useful for differentiating clinically relevant sensitization from cross-reactivity. By defining the relevant allergenic components, it improves diagnostic accuracy, refines risk assessment, and guides personalized recommendations on food avoidance and cofactor management. This approach in pediatric patients is particularly important, as it reduces the burden of unwarranted dietary restrictions and helps families focus on the true risk foods. Serum tryptase measurement after a reaction may support the diagnosis by confirming mast cell activation, although elevated levels are not always present in FDEIA [4].

In cases where results are inconclusive or unclear, controlled food-exercise challenge tests may be necessary, gradually combining the suspect food with physical activity. These tests must be conducted in specialized centers due to the high risk of severe reactions [5,31].

FDEIA treatment consists of preventive and acute-phase management strategies. Prevention involves patient education and behavioral modification, including avoiding the suspect food for at least 4 hours before and 1 hour after exercise, abstaining from NSAIDs and alcohol, and monitoring other cofactors. Maintaining a food-and-exercise diary can help identify recurring patterns [4,11].

In the event of a reaction, intramuscular adrenaline is the first-line treatment and should be always carried by the patient. Antihistamines and corticosteroids may be used as additional therapy but are not substitutes for adrenaline [11].

CONCLUSIONS AND FUTURE PERSPECTIVES

In recent years, research on FDEIA has significantly advanced our understanding of its pathophysiological mechanisms, particularly concerning the role of cofactors and allergen molecular components. Studies have demonstrated that the food-exercise combination alone

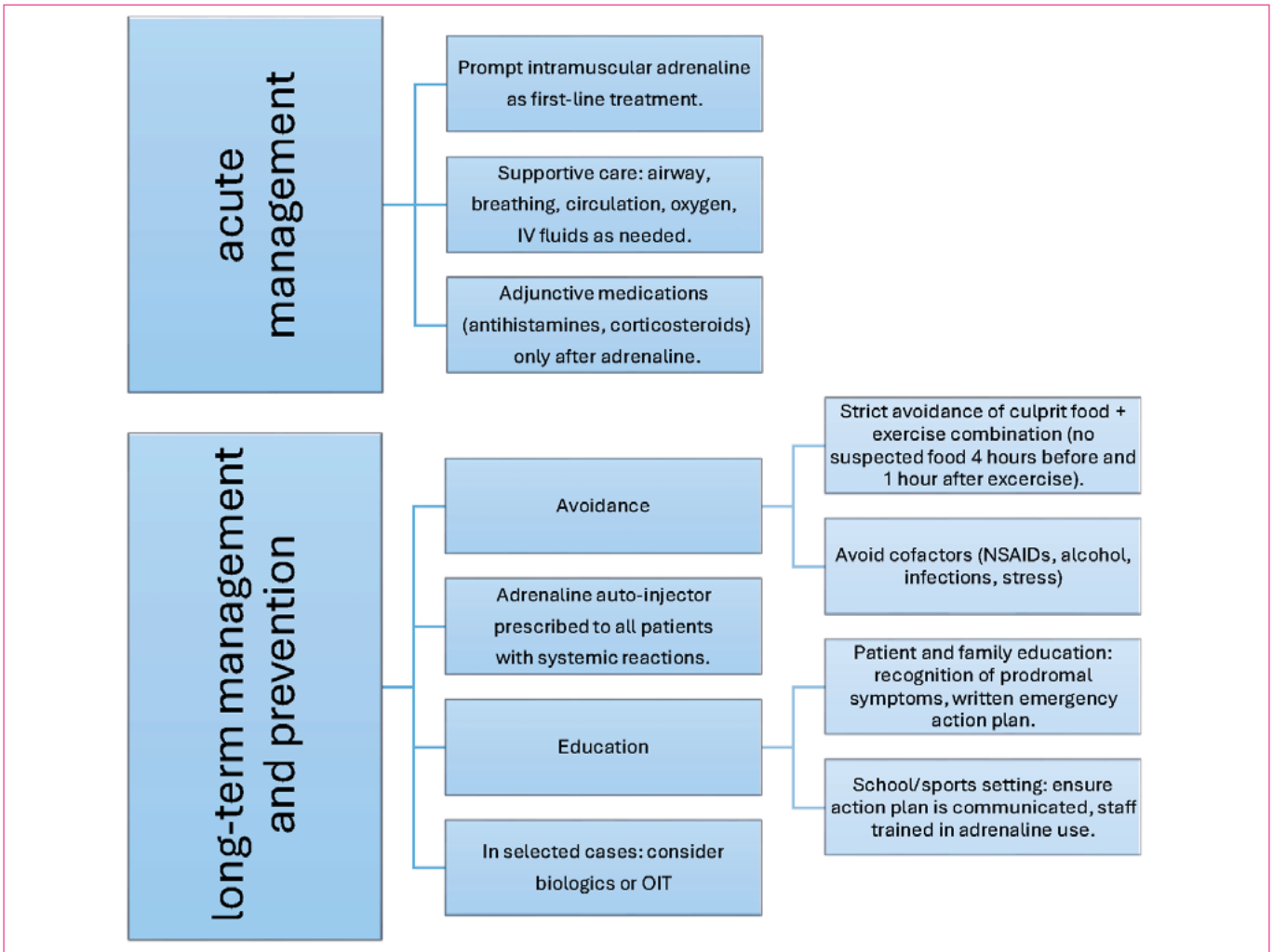


FIGURA 1. Summary of management strategies in patients with food-dependent exercise-induced anaphylaxis.

does not fully explain all reactions, emphasizing the importance of additional elements such as medications, alcohol, microbiota imbalances, and individual physiological conditions. Nonetheless, many aspects of FDEIA remain unclear, and there are still no universally accepted diagnostic criteria. Within this context, new therapeutic perspectives are emerging.

A rapidly evolving area in allergy treatment involves biological drugs. In food allergies, the FDA has approved omalizumab, a humanized IgG1 monoclonal antibody that selectively binds the Cε3 fragment of free IgE. In FDEIA patients, several cases have shown that omalizumab effectively prevents anaphylactic reactions [32-34]. There is also growing interest in dupilumab, an IgG4 monoclonal antibody that inhibits IL-4 and IL-13 signaling. Cases have been reported of patients with FDEIA who are protected from symptom onset during dupilumab treatment [35-37].

Additionally, numerous studies have evaluated oral immunotherapy (OIT) for food allergies. In selected patients, particularly those sensitized to LTPs and allergic to multiple fruits and vegetables - where avoidance is challenging - tolerance induction could be considered a rational intervention. However, cases of FDEIA onset during or after OIT have also been reported, necessitating careful patient selection and strategic implementation [38,39].

In conclusion, FDEIA is a complex clinical condition requiring an integrated and personalized approach. Promoting early diagnosis, accurate risk stratification, and proper management is essential to reduce the incidence of severe episodes and improve the quality of life for affected individuals.

Ethical consideration

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Conflicts of interest statement

The authors declare no conflicts of interest in relation to this publication.

Author's contributions

AZ conceived the manuscript, wrote the first draft, and finalized it. IB contributed to writing and finalizing the draft. PB conceptualized and supervised the project. All authors revised the manuscript and approved the final version.

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