УДК: 616-056.3-001.36:613.26/.29]-07-083.9-053.2 DOI: 10.31655/2307-3373-2026-25-3-51-63

ANAPHYLACTIC SHOCK IN CHILDREN CAUSED BY FOOD ALLERGENS: CURRENT APPROACHES TO DIAGNOSIS AND STANDARDS OF EMERGENCY CARE IN CLINICAL CASES

L. V. Besh[*,A,D,F], O. I. Matsyura[B,C,D,E], Z. L. Slyuzar[B,C,D,E], O. P. Borysiuk[B,C,D,E]

State Nonprofit Company "Danylo Halytsky Lviv National Medical University", Lviv, Ukraine

A — concept and design of the article; B — collection of clinical cases and literature review; C — analysis and interpretation of data; D — writing of the article; E — editing of the article; F — final approval of the article

Abstract. This article presents the current paradigm for the diagnosis and active management of patients with food allergies and anaphylaxis in pediatric practice. It analyzes the evolution of the WAO and EAACI 2025 clinical criteria, the specific "masks" of anaphylaxis in young children, and the elimination of strict time frames for symptom manifestation. A step-by-step algorithm for emergency care in accordance with the latest ERC 2025 recommendations (Pediatric Life Support), the principles of risk stratification for biphasic reactions, and the prospects for the use of nasal epinephrine are provided.

The aim of this study is to analyze two clinical cases of patients with anaphylaxis and to find opportunities for developing food tolerance, taking into account current algorithms.

Research materials and methods. Analysis of the patients` medical records and the results of clinical and laboratory tests.

Research Results. A 4-year-old girl had two episodes of anaphylaxis (at the ages of 11 months and 1 year and 3 months of age) following the consumption of a lysozyme-containing lozenge and an egg, respectively. After a prolonged adhehernce to the elimination diet and oral provocation tests, treatment using the egg ladder method was initiated. The dynamics showed a decrease in the levels of specific antibodies to egg components: ovomucoid (Gal d 1) from 3.53 to 0.2 kU/L, ovalbumin (Gal d 2) from 9.47 to 1.59 kU/L, and lysozyme (Gal d 4) from 9.9 to 1.81 kU/L. The child successfully began to tolerate eggs that had been thoroughly cooked.

A 2-year-and-3-month-old boy suffered two episodes of anaphylaxis (at the ages of 8 months and 1 year and 3 months) following the consumption of dairy products. The results of molecular diagnostics revealed high levels of specific IgE to the major protein components of cow's milk: α-lactalbumin (Bos d 4) — 37.45 kUA/L, β-lactoglobulin (Bos d 5) — 28.25 kUA/L, and casein (Bos d 6) — 32.45 kUA/L. Taking into account the patient's medical history, clinical presentation, and laboratory test results, it was recommended to completely eliminate milk and all dairy products for at least six months.

Conclusions. 1. Treating a patient with a history of food anaphylaxis allows for the identification of a "window of opportunity" to consider the reintroduction of the product, but this must be supported by an accurate diagnosis, supervision of an experienced team in a hospital setting, and only after an elimination period lasting at least 6 months. 2. Patients at high risk for anaphylaxis should be provided with a first-line medication — epinephrine — and given clear instructions on actions to take.

Key words: anaphylaxis, children, food allergy, molecular diagnostics, epinephrine.

1. Anaphylaxis in Modern Pediatrics: Prevalence, Triggers, and Scientific Debates

Anaphylaxis is a severe, potentially life-threatening generalized or systemic hypersensitivity reaction. It is characterized by a rapid onset with critical impairments of respiratory and circulatory functions and is typically associated with manifestations on the skin and mucous membranes [1, 2, 3]. However, in clinical practice, anaphylactic shock can develop even without skin symptoms [1, 3]. This pathological condition has an acute course with severe hemodynamic disturbances, leading to a systemic circulatory failure and hypoxia of the vital organs [1, 3, 4].

The pathogenesis of anaphylaxis is based on the rapid activation of mast cells (tissue basophils), primarily through IgE-mediated pathways, accompanied by a massive release of biologically active mediators, particularly of tryptase and histamine. Basophils may also contribute to the development of anaphylaxis. The lifetime prevalence of anaphylaxis ranges from 0.3 % to 5.1 %, and its incidence is steadily increasing worldwide among both adult and pediatric populations, a trend linked to the rapid rise in the overall prevalence of allergic pathology. The results of recent meta-analyses indicate that the global incidence rate is approximately 46 cases per 100,000 people per year, with an annual

increase in the number of new cases of anaphylaxis of about 7.4 % [1, 3, 5].

Today, scientific discussions on anaphylaxis are focused on a number of pressing issues [6]:

- Risk of anaphylaxis recurrence;
- An optimal algorithm for the dynamic monitoring of patients who have experienced an acute condition;
- The use of intranasal epinephrine as an effective needle-free alternative;
- Characteristics of the microbiome in patients prone to acute, life-threatening systemic reactions;
- The mental state of patients who have experienced anaphylaxis and the need for psychological intervention to address associated risks;
- The use of targeted biological therapy in patients with food allergies to reduce the risk of anaphylaxis during oral immunotherapy;
- Concomitant mastocytosis as a factor that significantly increases the risk of severe, life-threatening anaphylaxis development;
- A reassessment of the role of serum tryptase as the primary laboratory criterion for verifying and assessing the risk of anaphylaxis.

In pediatric practice, the main triggers of anaphylaxis are foods (40–45 %), medications (30–35 %), stinging insect venom and latex (10–12 %), and other factors (10–15 %) [7]. Among the foods that most commonly cause anaphylaxis in children are cow's milk, chicken eggs, soy, wheat, peanuts, tree nuts, and seafood [2, 8]. According to statistics from FoodAllergy.org, peanuts are the most common cause of fatalities due to food-induced anaphylaxis in the general population.

2. "Masked" and multi-organ manifestations of food anaphylaxis in young children

Food anaphylaxis in children may present with nonspecific symptoms, including persistent inconsolable crying, drowsiness, watery eyes, a sore throat, severe anxiety, numbness in the limbs, as well as difficulty swallowing and speaking. For this reason, it is extremely difficult to identify the etiological triggers in young children, since taking a medical history and subjectively assessing symptoms is less informative, and clinical symptoms are often transient or nonspecific. In clinical practice, a severity classification is used that distinguishes three degrees of anaphylactic reaction: mild, moderate, and severe, involving various target organs (the skin, the gastrointestinal tract, and the respiratory, cardiovascular, and nervous systems) [1, 3, 4, 9].

Most young children exhibit a variety of dermatological symptoms: urticarial rash, itching, generalized erythema, angioedema, as well as indirect signs such as difficulty swallowing or constant licking of the lips [9]. Gastrointestinal symptoms range from mild itchiness or a tingling sensation in the mouth and nausea to severe cramping abdominal pain, repeated vomiting, and diarrhea. Respiratory disorders may present as nasal congestion, rhinorrhea, hoarseness, bronchospasm, cyanosis, and respiratory arrest [2, 9]. Cardiovascular manifestations include tachycardia, bradycardia, hypotension, vascular collapse, arrhythmias, and cardiac arrest. Neurological symptoms include changes in activity level, lethargy, disorientation (or delirium), fear of death, confusion, or complete loss of consciousness [1, 4].

According to statistics, in cases of anaphylaxis in children, skin symptoms are reported in 80–90 % of cases, respiratory symptoms in 70 %, gastrointestinal symptoms in 30–45 %, cardiovascular symptoms in 10–45 %, and neurological symptoms in 10–15 % [4, 8]. Thus, early acute signs of anaphylaxis may include nasal discharge, itching of the eyes, lips, or ears, and facial swelling. Severe reactions are associated with the development of bronchospasm and laryngospasm, gastrointestinal symptoms, and progressive hypotension [1, 4].

Clinical signs of anaphylaxis usually develop within a few minutes to two hours after exposure to an allergen, and in cases of acute IgE-mediated food allergy in children, symptoms typically appear as early as 15–30 minutes after consumption of the causative food.

However, it was precisely this strict time-frame approach that became one of the factors leading to the revision of international protocols.

3. The evolution of the anaphylaxis definition and diagnostic criteria

Current data shows significant differences in the definitions of anaphylaxis, which have long posed challenges for clinicians and complicated the standardization of treatment approaches [2, 3, 9-14]. The main drawback of many classical definitions was the strict requirement that multiple organ systems be affected simultaneously. However, current clinical practice and comprehensive reviews by leading specialized organizations (WAO, EAACI, AAAAI) show that critical and life-threatening symptoms can manifest only within a single organ system [15-17]. The view that anaphylaxis

ISSN 2307-3373

is solely a multi-organ process has been recognized as erroneous and dangerous, as it disorients physicians and leads to a fatal delay in providing care.

Another important aspect is the perception of anaphylaxis as a potentially fatal reaction. Statistical data indicates that fatal and near-fatal cases are relatively rare, even in the absence of timely treatment [18-22]. Since it is impossible to predict the rate and nature of symptom progression in a specific patient [21], a fundamental principle of emergency care is the immediate intramuscular administration of epinephrine at the first reasonable suspicion of anaphylaxis to preventively reduce the risk of lethality [22].

Systemic glucocorticoids (GCs) and antihistamines have been reclassified as strictly adjunctive therapies due to their significant pharmacodynamic limitations. Antihistamines block only H1/H2 receptors, affecting only local skin symptoms, whereas the clinical effect of corticosteroids is delayed (at least 4–6 hours), making them ineffective for the emergency treatment of acute airway edema or progressive vascular collapse. Furthermore, current body of evidence has refuted the claim that GCs can prevent the development of biphasic reactions. Thus, the initial use of glucocorticoids or antihistamines–based on the mistaken perception that they are a "safer" alternative–and the delay in administering epinephrine until the patient's condition has critically deteriorated are considered a serious tactical error. In clinical practice, this therapeutic strategy most often precedes the development of irreversible vascular collapse and asystole in cases of anaphylaxis.

Therefore, epinephrine remains the only pathogenetically justified first-line treatment; its administration in the first minutes of anaphylaxis provides an immediate effect due to the synergistic activation of α1-adrenoreceptors (vasoconstriction, hemodynamic stabilization, reduction of tissue edema) and β_2-adrenoreceptors (potent bronchodilation and inhibition of further mast cell degranulation with the release of inflammatory mediators).

The evolution of diagnostic approaches has made it possible to address several critical "blind spots" inherent in the previous NIAID/FAAN criteria [14, 23]. Firstly, it has been established that a significant proportion of fatal reactions caused by food triggers, medications, or insect stings begin in isolation with respiratory or cardiovascular disturbances [15-17], which now serves as a direct basis for the diagnosis of anaphylaxis. Secondly, a clear classification of systemic manifestations by severity has been established: the development of generalized skin symptoms (urticaria, angioedema) provided that the airways remain unobstructed, without bronchospasm, and with stable age-appropriate blood pressure and pulse rates is classified as acute generalized urticaria. This nosology does not pose an immediate threat to the patient's life and does not require urgent administration of epinephrine, thereby avoiding unnecessary polypharmacy [2].

Thirdly, modern diagnostic protocols take into account the specific characteristics of triggers with delayed onset, particularly alpha-gal allergy syndrome (IgE-mediated hypersensitivity to the oligosaccharide galactose-α-1,3-galactose) [24]. In this pathological condition, the first symptoms appear only 3–6 hours after consuming mammalian meat (due to the prolonged digestion of lipids and transport of glycoproteins); however, following the initial onset, the reaction is characterized by an aggressive course and rapid progression to a vascular collapse. Verification of this mechanism prompted the WAO Committee to remove the strict time limits ("from a few minutes to a few hours") from the updated criteria.

Fourthly, in order to eliminate geographical and conceptual discrepancies in global practice [25, 26] the ambiguous term "persistent gastrointestinal symptoms" was replaced with the definition "severe gastrointestinal symptoms," which includes severe cramping abdominal pain, repeated vomiting, etc. According to current criteria [1, 3], isolated severe gastrointestinal manifestations after confirmed exposure to a known systemic non-food allergen (insect bite, administration of medications) are considered equivalent to anaphylaxis requiring immediate administration of epinephrine, even in the absence of cutaneous markers.

Finally, the most dangerous factor delaying the verification of a diagnosis is the stereotype that skin symptoms are mandatory. It has been clinically proven that in 10–20 % of anaphylaxis cases (especially in fatal episodes involving fulminant shock or asphyxia), skin manifestations are completely absent [16, 17].

Usually, the absence of these skin markers confuses clinicians, leading to misdiagnosis and, as a result, inappropriate treatment strategies (for example, an incorrect diagnosis of an isolated severe asthma attack or acute heart failure). The official recognition that anaphylaxis can occur without skin involvement served as the basis for a fundamental change in the formulation of diagnostic criteria, stipulating that, in the presence of a clear link to an allergen and the rapid onset of respiratory or hemodynamic collapse, a diagnosis of

anaphylaxis should be given immediately, regardless of the skin condition.

The WAO Anaphylaxis Committee has proposed a new, more precise definition. Anaphylaxis is a severe systemic hypersensitivity reaction that typically manifests rapidly and can be life-threatening. In recognition of this, the focus of diagnosis has shifted to the standardized ABC triad of vital criteria: airway patency (Airway–laryngeal or tongue edema), adequate breathing (Breathing–severe bronchospasm), and stable circulation (Circulation–arterial hypotension) [23]. In this context, the expert group of the WAO Special Committee made an important conceptual breakthrough by establishing that the severity of anaphylaxis is determined not by the number of organ systems involved in the pathological process, but by the immediate threat to vital functions. It also debunked the widespread clinical myth that the development of obvious signs of shock (collapse, loss of consciousness) is mandatory for verifying a critical condition. Researchers have demonstrated that a terminal life-threatening condition–specifically, complete asphyxia resulting from fulminant laryngeal edema–can occur even when systemic blood pressure remains normal or within the normal range. This clarification allows clinicians to diagnose severe anaphylaxis and initiate epinephrine administration in the early stages, without wasting time expecting hemodynamic parameters to deteriorate.

To simplify the practical application of the criteria, the World Allergy Organization (WAO) introduced unified diagnostic criteria in 2020 [3]. According to these criteria, anaphylaxis was considered highly probable if at least one of two criteria was present simultaneously:

Table 1. Updated 2025 GA²LEN and WAO Consensus Criteria for the Diagnosis of Anaphylaxis

The probability of anaphylaxis is high if at least one of the three criteria listed below is met	
Criterion 1: **allergen UNKNOWN (anaphylaxis without a clear link to a specific allergen)**	**Sudden onset of the disease** (ranging from a few minutes to several hours) with inevitable **involvement of the skin and/or mucous membranes** (hives, diffuse redness, a sensation of heat, erythema, swelling of the face, lips, tongue, or oropharynx, a feeling of tightness in the throat, and difficulty swallowing). *In infants: marbled skin, repeated lip licking.* ***+ at least one of the following symptoms***: 1. **Respiratory System:** wheezing, shortness of breath (dyspnea), increased respiratory effort, hypoxemia, cough; *laryngeal symptoms:* stridor, voice changes. *In infants – a hoarse cry).* 2. **Cardiovascular System:** arterial hypotension, loss of consciousness, dizziness, unexplained changes in mental status. *In infants: persistent, unexplained tachycardia.*
Criterion 2: **PROBABLE or known exposure to an allergen**	**An acute, sudden onset accompanied by the simultaneous, rapid involvement of at least two of the following systems**: 1. **Skin and mucous membranes:** hives, diffuse redness, a sensation of heat, erythema, swelling of the face, lips, tongue, or throat, a feeling of tightness in the throat, difficulty swallowing. *In infants: marbled skin, repeated lip licking.* 2. **Respiratory system:** wheezing, shortness of breath (dyspnea), increased respiratory effort, hypoxemia, cough; *laryngeal symptoms*: stridor, voice changes. *In infants – a hoarse cry).* 3. **Cardiovascular System:** arterial hypotension, loss of consciousness, dizziness, unexplained changes in mental status. *In infants: persistent, unexplained tachycardia.* 4. **Gastrointestinal tract:** intense, cramp-like abdominal pain, repeated vomiting, and diarrhea.
Criterion 3: **KNOWN exposure to an allergen**	**The manifestation of any one of the following systemic disorders (regardless of the time elapsed since exposure to the allergen):** **Respiratory system:** wheezing, shortness of breath (dyspnea), increased respiratory effort, hypoxemia, cough; *laryngeal symptoms:* stridor, voice changes following exposure to a non-inhaled allergen (such as food allergen, medication, or an insect bite). *In infants – a hoarse cry.* 2. **Cardiovascular system:** hypotension, loss of consciousness, dizziness, unexplained changes in mental status. *In infants: persistent, unexplained tachycardia.*

ISSN 2307-3373

Table 2. A Comparative Analysis of the WAO (2020) and GA²LEN (2025) Consensus Criteria

Indicator	WAO (2020) Criteria	New GA²LEN tool (2025)
Diagnostic Framework	**2 rigid points** (an attempt for maximum simplicity)	**3 flexible clinical scenarios** (depending on exposure to the trigger)
Scenario without known contact	Requires a combination of skin/mucous membrane **AND** (breathing **OR** blood pressure **OR** severe gastrointestinal symptom).	Requires a combination of skin/mucous membrane AND (breathing **OR** cardiovascular disorders)
Status of severe gastrointestinal symptoms	Considered an additional manifestation (primarily for non-food allergens).	They became a **fully-fledged, equivalent criterion** in Scenario 2 (even for food triggers)
Known contact with an allergen	May cause isolated respiratory **AND/OR** cardiovascular disorders.	Clearly states: the sudden onset of an **isolated** respiratory or cardiovascular disorder is sufficient
Clinical focus	Aimed at standardizing the definition of anaphylaxis.	Aimed at **minimizing hypodiagnosis** and facilitating rapid decisions regarding the administration of epinephrine

1. Typical skin symptoms (generalized urticaria, angioedema) in combination with significant abnormalities in at least one other organ system (respiratory, cardiovascular, or gastrointestinal);
2. Rapid onset of isolated respiratory and/or cardiovascular abnormalities upon exposure to a known or suspected allergen for this patient.

However, this simplification of the criteria had a downside, as it often led to underdiagnosis of anaphylaxis in emergency care settings. The rigid two-component protocol complicated the diagnosis of anaphylaxis, causing physicians to have doubts when the condition presented in a non-typical pattern.

The new clinical guidance Anaphylaxis Clinical Support Tool (GA²LEN, 2025) successfully addresses this issue [1]. Instead of a standardized definition, it offers a flexible, tactical approach, categorizing patients into three distinct clinical scenarios based on the presence and certainty of allergen exposure (Table 1)

The updated diagnostic approach requires that even early, systemic reactions that are just beginning to develop during allergen immunotherapy (in particular, subcutaneous administration of allergens) be considered and treated as anaphylaxis.

Despite the expansion of diagnostic criteria, differential diagnosis remains critically important in clinical practice. The symptom complex of anaphylaxis must be carefully distinguished from other acute conditions that present with a similar clinical picture, in particular severe exacerbations of bronchial asthma, isolated localized angioedema, vasovagal syncope (fainting), as well as acute anxiety attacks and panic attacks.

5. Algorithm for Providing Emergency Care for Anaphylaxis in Children (based on the official 2025 guidelines of the European Resuscitation Council (ERC))

In pediatric practice, immediate recognition and urgent treatment of anaphylaxis are critical to preventing asphyxia and cardiac arrest. The current European Resuscitation Council (ERC 2025) protocol [27] outlines the following sequence of actions:

Step 1. Initial assessment and elimination of the trigger

Immediate recognition: the condition is typically characterized by an acute onset and rapid progression of cutaneous, respiratory, cardiovascular, and/or severe gastrointestinal symptoms.

Elimination of the allergen: if possible, administration of or contact with any potential allergen must be immediately stopped.

Step 2. Emergency Administration of Epinephrine (First-Line Treatment)

Epinephrine (1 mg/mL) must be immediately administered intramuscularly (IM) into the anterolateral surface of the middle third of the thigh.

Dosage – 0,01 mg/kg (maximum single dose 0,5 mg).

Quick dosage guide by age (if weight is unknown):

1–5 year olds: **0,15 mg IM** (or an autoinjector with the appropriate dosage);

6–12 year olds: 0,3 mg IM or an autoinjector with the appropriate dosage);

over 12 years of age: 0,5 mg IM.

Repeated administration of epinephrine. If symptoms persist, repeat the specified dose of epinephrine intramuscularly every 5 minutes.

The official approval by the U.S. Food and Drug Administration (FDA) and the European Medicines Agency (EMA) of the first nasal epinephrine spray was a true breakthrough. The nasal form eliminates the fear of injections among children, their parents, and teachers, which dramatically simplifies the administration of first aid in critical situations. Studies have confirmed that the bioavailability of epinephrine in spray form and its onset of action are equivalent to intramuscular injection, and that absorption remains effective even in the presence of concomitant rhinitis. Intranasal epinephrine solution is recommended for children at a dose of one spray in each nostril: 2 mg/0.1 mL for children weighing more than 30 kg, and 1 mg/0.1 mL for children weighing between 15 kg and 30 kg.

According to current international standards [1, 27, 28], there is no strict limit on the total number of intramuscular doses of epinephrine. The remedy is administered as many times as the situation requires to save the patient's life. However, the recommendations mentioned above clearly define the critical points at which treatment strategies should be adjusted. Thus, if the expected therapeutic effect is not achieved after the administration of 2 *intramuscular doses* of epinephrine, the condition is classified as *refractory anaphylaxis, which, in particular, calls for* a switch to an *intravenous titrated infusion of epinephrine. However*, this procedure is performed exclusively in an intensive care unit under continuous cardiac monitoring (ECG, blood pressure, pulse oximetry) due to the high risk of arrhythmia and hemodynamic instability.

Step 3. Positioning and Oxygen

Assess using the ABCDE criteria and continuously monitor vital signs.

Optimize body positioning. Place the child in a position appropriate for the predominant set of symptoms.

If there are signs of shock (the child is pale, impaired consciousness/ lost consciousness, low blood pressure), place the child in a strictly horizontal supine position with the legs elevated. This positioning is critical for preventing the development of "empty heart syndrome," since distributive shock is characterized by massive vasodilation and redistribution of blood. The patient must not suddenly stand up or sit down, as this can trigger an immediate and irreversible vascular collapse.

If signs of respiratory failure are predominant (wheezing, stridor, cyanosis of the lips), the patient should be placed in a sitting or semi-sitting position (to facilitate chest excursion and reduce shortness of breath).

Oxygen therapy. The administration of 100 % oxygen should be prescribed for all children exhibiting signs of respiratory failure, as well as for patients who have required more than one dose of epinephrine.

Step 4. Ensuring airway patency and vascular access

Airway management. Early tracheal intubation should be considered in cases of progressive respiratory distress or if there is a risk of laryngeal or tongue edema. Since maintaining airway patency under such conditions is technically challenging, it is essential to immediately call a qualified pediatric anaesthesiologist experienced in complex intubation techniques.

Volemic support. To catheterize a peripheral vein or establish intraosseous access.

For hemodynamic stabilization and management of shock, administer a bolus of crystalloid solutions at a rate of 10 mL/kg.

Step 5. Additional therapy

To relieve symptoms of bronchospasm, short-acting inhaled β2-agonists (salbutamol) should be used as adjunctive therapy to the baseline intramuscular administration of epinephrine.

Expert assistance. If the symptoms persist and more than two doses of epinephrine are required, immediately call (consult) a pediatric intensivist.

Step 6. Medical follow-up, second-line therapy, diagnosis

Following the complete resolution of acute symptoms, the duration of inpatient monitoring is no longer routinely 24 hours, but is determined by an individual risk stratification for the development of biphasic reactions.

Low-risk group: if symptoms resolved quickly and completely after administration of the first (single) dose of epinephrine, the observation and monitoring period may be shortened to 2–4 hours.

High-risk group: patients with severe anaphylaxis accompanied by hypotension, requiring multiple doses of epinephrine, as well as those with concomitant severe bronchial asthma or mastocytosis, require prolonged monitoring (6–12+ hours).

Once the patient's condition has stabilized, second-line medications may be used. Antihistamines are indicated exclusively for the treatment of isolated skin symptoms such as itching and urticarial rash. Glucocorticosteroids are recommended for patients with concomitant bronchial asthma.

ISSN 2307-3373

During the acute phase, it is recommended to draw blood in order to measure serum tryptase levels for the purpose of retrospectively confirming mast cell degranulation (ideally within 1–2 hours of symptom onset).

6. A new era in the treatment of food allergies: A comprehensive approach based on the updated EAACI standards

The current strategy for managing patients with IgE-mediated food allergy has undergone a fundamental paradigm shift. While the traditional approach involved passively waiting for spontaneous remission while following a strict elimination diet, the updated guidelines from the European Academy of Allergy and Clinical Immunology (EAACI, 2025) call for a shift toward active management [29].

The final recommendations of the working group systematize clinical approaches in five key areas; the fundamental differences between these approaches and previous guidelines are presented in Table 3.

Clinical Glossary EAACI Guidelines 2025

Desensitization is a temporary increase in the sensitivity threshold, during which a patient can safely consume a certain amount of an allergen exclusively while undergoing therapy (once treatment is discontinued, the protective effect disappears and sensitivity returns).

Remission, or persistent lack of reactivity, is the ability to safely consume a specified amount of a product after discontinuing active treatment for a certain period of time (sustained clinical tolerance following discontinuation of therapy).

Tolerance is the body's persistent ability to consume food without developing any symptoms, regardless of the amount or frequency with which the food is consumed (full recovery).

Detailed description of key management focus areas according to EAACI 2025

Dietary management of patients

The cornerstone of treatment remains the rational limitation of exposure to the causative factor: experts have issued a strong recommendation (despite the low certainty of the evidence) that patients must avoid a specific food allergen or the form of that allergen to which they have a confirmed clinical reaction. At the same time, a strict inclusive approach to diet planning has been adopted. Based on moderate levels of evidence, a strong recommendation is made regarding the need for long-term and regular consumption of all other foods that the patient can tolerate. This helps prevent iatrogenic nutritional deficiency and support natural immune tolerance.

Table 3. A paradigm shift in food allergy management: a comparative analysis

Area	Traditional Approach	Modern Concept (EAACI 2025)
Patient management strategy	A passive "watch-and-wait" approach, with monitoring of spontaneous resolution over time.	Active management: early prophylactic introduction of allergens into the diet, individualized dietary algorithms, and active immunomodulatory interventions.
Philosophy of dietary therapy	Total elimination based on the "just in case" principle. Strict diets that led to nutritional deficiencies and developmental delays in children.	Maximum range and inclusivity: foods that the patient can actually tolerate are kept in the diet. Introduction of heat-treated foods (baked milk and egg products).
Specific immunotherapy	Lack of etiological treatment. Treatment was limited to managing symptoms in cases of accidental exposure.	Allergen-specific immunotherapy (ASIT): oral (POIT), sublingual (SLIT), and epicutaneous (EPIT) therapy to increase the sensitivity threshold.
Pharmacotherapy	Extensive post-factum symptomatic treatment (routine and often unjustified prescribing of antihistamines and systemic steroids).	**Target biological therapy**: the use of omalizumab (anti-IgE) for desensitization of patients with multiple allergies, minimization of the risk of anaphylaxis, and preventive protection.
Psychological support	The psychological well-being of the child and the parents was either ignored or considered secondary.	**Official recognition of allergy-related anxiety** as a factor contributing to severe distress for the entire family. It is recommended to involve clinical psychologists and utilize cognitive-behavioral therapy (CBT).

Psychological support

For the first time, the guidelines devote specific attention to the mental health of families. Based on low certainty of evidence, a conditional recommendation has been formulated regarding the advisability of involving qualified healthcare professionals to provide psychological support to certain categories of patients and their parents (caregivers) with the aim of reducing allergy-related anxiety and improving quality of life.

Emergency care organization and treatment plans

To minimize the risk of fatal consequences from accidental contact with a trigger, the EAACI insists on the implementation of clear safety protocols. The following are mandated as strong recommendations (with low certainty of evidence):

1. Development of a personalized written management plan for each patient.
2. Mandatory prescription and constant carrying of epinephrine autoinjectors (EAIs) by individuals with a documented risk of anaphylaxis.
3. Conducting structured, comprehensive trainining for patients, their parents (or legal guardians), as well as staff at children's institutions (caregivers, teachers) on how to recognize the early signs of anaphylaxis and the proper use of EIs.

Food allergen-specific immunotherapy (ASIT)

All types of desensitization carry a potential risk of systemic side effects; therefore, the working group issued a strong recommendation (level of evidence: low) that any food ASIT protocols be conducted exclusively under the supervision of an experienced clinical team trained in providing emergency care for anaphylaxis.

Peanut allergy: has the strongest evidence base. Peanut oral immunotherapy (POIT) for children and adolescents has received a strong recommendation based on high certainty of evidence. The epicutaneous method (EPIT) and the sublingual variant (SLIT) are proposed as alternative desensitization approaches at the level of conditional recommendations (certainty of evidence: high and moderate, respectively).

Allergies to chicken eggs and cow's milk: oral ASIT for eggs or milk is recommended as a conditional recommendation (low quality of evidence), primarily for children over 4 years of age and adolescents with persistent forms of the disease (given the high frequency of spontaneous development of natural tolerance in early childhood).

Biologic medications in Allergy Medicine

The use of monoclonal antibodies has marked a new step forward in the management of this condition. Based on moderate levels of evidence, experts have issued a conditional recommendation regarding the use of omalizumab (anti-IgE therapy) to successfully desensitize patients to one or more food allergens simultaneously.

Based on the results of the groundbreaking OUTMATCH study led by R. Wood and co-authors [30], omalizumab as monotherapy enables patients with multiple food allergies to safely tolerate significant doses of peanuts, milk, eggs, and cashews. Given its high clinical efficacy, this drug has already been officially approved by regulatory agencies (including the FDA) for the treatment of food allergies in children aged 1 year and older.

Thus, the integration of allergen-specific immunotherapy, targeted biologics, and mandatory psychological support within the EAACI 2025 strategy facilitates a shift from passive patient isolation to the controlled development of immune tolerance and an improvement in the patient's quality of life.

ANALYSIS OF CLINICAL CASES

Clinical Case No. 1

A girl, 4 years old.

She was born following a full-term pregnancy in a normal delivery.

Table 4. Criteria for prescribing epinephrine auto-injectors (AIA) for IgE-mediated food allergy (according to current EAACI guidelines)

Absolute indications	Relative indications
Previous epidodes of food anaphylaxis Concomitant unstable or moderate/severe persistent bronchial asthma Concomitant systemic mastocytosis	An allergic reaction to foods that are commonly associated with anaphylaxis An allergic reaction to minimal (trace) amounts of food Adolescence or young adulthood (due to the high risk of dietary violations) Geographical distance from medical care or extended trips abroad Patients currently undergoing ASIT for food allergy

ISSN 2307-3373

She was breastfed for 8 months, during that time the mother did not follow any specific diet.

There is an aggravated family history: the mother suffers from hay fever, and the father is allergic to cats.

Medical history: The girl has had symptoms of atopic dermatitis since birth (dry skin, recurrent rashes of unknown origin on her body, which resolved after a short course of antihistamines).

At **11 months of age, the child experienced anaphylaxis for the first time in her life** after consuming a crushed lozenge containing lysozyme (generalized rash over the body, itching, swelling around the eyes and lips, vomiting). The emergency medical team administered dexamethasone and chloropyramine. Up to that point, eggs had never been introduced into the child's diet.

Introducing crushed lozenges to a young child carries several risks: legal liability for off-label use of the medication, the risk of aspiration, and the individual risk of a severe allergic reaction.

After their child experienced an anaphylactic reaction, the parents followed an egg-free elimination diet and kept a first-aid kit on hand for emergency treatment.

When the girl was 1 year and 3 months old, she experienced a second anaphylactic reaction (severe rhinoconjunctivitis, vomiting, restlessness) after accidentally ingesting a lightly fried egg (one teaspoon). Her mother administered epinephrine using an auto-injector. The ambulance crew administered ondansetron and prescribed desloratadine.

The allergist provided the following long-term recommendations:

1. A diet that completely and strictly excludes eggs and egg products.
2. Ensuring the availability of two epinephrine auto-injectors. During the visit, the parents received repeated training on how to recognize symptoms of anaphylaxis in their child, as well as the rules and procedure for administering emergency treatment.
3. Vitamin D
4. Skin moisturizing.

Oral provocation test with an egg

At age 2, an oral egg provocation test was performed in a hospital setting. The child asymptomatically consumed muffins containing egg (baked at 180°C for 30 minutes), reaching a cumulative dose of 1.5 g of egg protein.

Thus, after 9 months of complete elimination of eggs from the diet, the process of developing oral tolerance to eggs was successfully initiated by using the food ladder method at home.

After receiving the results of the oral food provocation test, the mother began introducing foods containing a minimal amount of egg on a daily basis, but only those that had been thoroughly cooked (muffins, cookies, crackers, egg noodles) — **Step 1 of the egg ladder**. At the same time, she strictly avoided giving the child eggs as a separate food item (boiled or fried). This stage lasted 4 months.

Over the next 4 months, the duration of heat treatment was reduced, and the number of egg-based dishes (pancakes, crepes, waffles, cutlets, meatballs) was gradually increased – **Step 2 of the egg ladder**.

Introduction of well-cooked egg dishes – **Step 3 of the egg ladder**. For 5 months, the girl ate hard-boiled eggs (15 minutes), toast, or vegetables baked or fried in an egg-and-milk mixture or egg batter.

The consumption of eggs after light heat treatment is **step 4 of the egg ladder**. At this stage, the patient is currently consuming omelets, scrambled eggs, custard, and boiled egg sauce (10 minutes). This stage allows for the consumption of a sufficient amount of egg white, but only when cooked, and includes a warning against including raw forms of the product in the diet.

Step 5 of the egg ladder – consuming raw or nearly raw egg components–was something that mom deliberately avoided.

At 3 years and 10 months of age, the girl developed mild acute urticaria after consuming a tablespoon of minimally cooked eggs. For breakfast, her father prepared 8 egg whites for himself, cooked for 1–2 minutes, which he sometimes eats after exercising. After taking antihistamines, the symptoms regressed in 45 minutes.

The girl underwent molecular testing using the ImmunoCAP method three times to determine specific IgE levels to egg components.

Ovomucoid (Gal d 1) is the primary and most aggressive allergen in chicken egg white. The level of specific antibodies decreased from 3.53 to 0.2 kU/L.

Dynamics of ovalbumin (Gal d 2) from 9.47 to 1.59 kU/L, and lysozyme (Gal d 4) from 9.9 to 1.81 kU/L.

This clinical case demonstrates successful treatment and the development of egg tolerance in a child with a medical history of two episodes of anaphylaxis.

Clinical Case No. 2

A boy, 2 years and 3 months old.

He was born of his mother's first pregnancy and first delivery. The pregnancy, delivery, and neonatal period proceeded without complications.

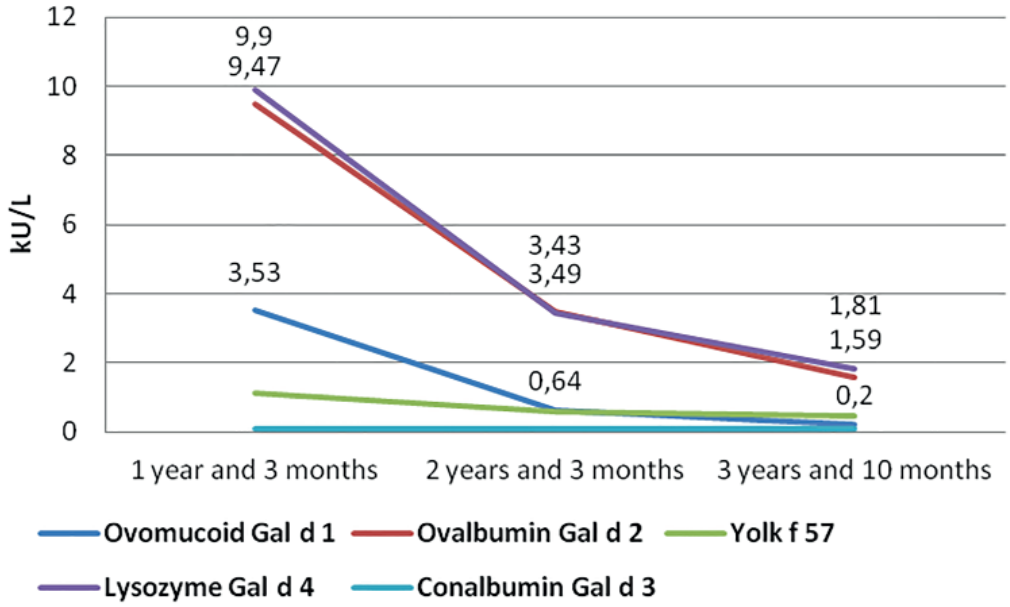

Figure 1. Shows the dynamics of specific IgE (sIgE) levels (molecular components) for egg in a child with a history of anaphylaxis during allergy treatment based on the food ladder method.

There is a family history of allergies: the mother has been diagnosed with hay fever, and the father with chronic urticaria.

The first signs of an allergic condition appeared at 2 months of age following consumption of a fermented milk formula and were characterized by the appearance of maculopapular rashes on the skin. Based on the clinical presentation, the family doctor diagnosed an allergy to cow's milk proteins. The child was recommended to be fed a formula based on extensively hydrolyzed whey proteins for six months, and a short course of treatment with antihistamines was also prescribed.

At 8 months of age, an acute systemic allergic reaction developed approximately 7 minutes after consuming one teaspoon of yogurt. The clinical presentation included generalized urticaria and angioedema, repeated vomiting, and respiratory symptoms such as a dry cough and shortness of breath. Upon arrival, **the emergency medical team diagnosed anaphylactic shock** and administered emergency treatment, which included the administration of epinephrine, ondansetron, and salbutamol.

Another episode of anaphylaxis occurred at 1 year and 3 months of age following the consumption of milk chocolate. The reaction was accompanied by a combination of cutaneous, gastrointestinal, and respiratory symptoms. Before medical personnel arrived, the mother administered epinephrine to the child on her own.

As part of a follow-up allergy evaluation, molecular diagnostics were performed using the ALEX-3 test. High levels of specific IgE to the major protein components of cow's milk were detected: α-lactalbumin (Bos d 4) – 37.45 kUA/L, β-lactoglobulin (Bos d 5) – 28.25 kUA/L, and casein (Bos d 6) – 32.45 kUA/L.

Based on the patient's medical history, clinical presentation, and laboratory test results, the following recommendations were provided to the parents:

1. Complete exclusion of milk and all milk-containing products from the child's diet for at least six months, followed by a reassessment of the child's condition and a review of the management strategy. At this stage, oral challenge tests are contraindicated.
2. Always carrying two epinephrine auto-injectors. Parents have been retrained on the early recognition of anaphylaxis symptoms, the algorithm for providing emergency care, the technique for using an auto-injector, and the administration of antihistamines

Thus, treating a patient with a medical history of food anaphylaxis allows for the identification of a "window of opportunity" to reconsider reintroducing the food, but this must be supported by an accurate diagnosis, supervision by an experienced team in an inpatient setting, and only after an elimination period lasting at least 6 months.

Conclusions and perspectives for future development

Based on the analysis of current international consensus and evidence-based data, several key points can be made.

Anaphylaxis remains a purely clinical diagnosis. Detecting elevated levels of tryptase in blood plasma

ISSN 2307-3373

and histamine in urine allows for retrospective laboratory confirmation of this condition; however, such tests should under no circumstances delay the initiation of treatment, as the results are not available quickly. The first step in emergency management at the slightest suspicion of anaphylaxis is the immediate administration of epinephrine.

A long-term monitoring of patients (long-term management) should focus on accurately identifying triggers by taking a detailed medical history, specific IgE testing, skin prick tests, or provocation tests, although up to 10 % of anaphylaxis cases remain idiopathic. After stabilization, each patient (or parents) should receive a written, individualized action plan and have constant access to epinephrine autoinjectors or nasal sprays for self-administration.

At this stage, there remains an urgent need to ensure global access to epinephrine delivery systems, standardize severity assessment criteria, improve medical education, and integrate digital health tools. Further research into biomarkers, genetic profiles, and cofactors of anaphylaxis is a priority area that will help optimize the diagnosis, prevention, and dynamic monitoring of this life-threatening condition.

АНАФІЛАКТИЧНИЙ ШОК У ДІТЕЙ, СПРИЧИНЕНИЙ ХАРЧОВИМИ АЛЕРГЕНАМИ: СУЧАСНІ ПІДХОДИ ДО ДІАГНОСТИКИ ТА СТАНДАРТИ НЕВІДКЛАДНОЇ ДОПОМОГИ В КЛІНІЧНИХ ВИПАДКАХ

Л. В. Беш, О. І. Мацюра, З. Л. Слюзар О. П. Борисюк

ДНТ «Львівський національний медичний університет імені Данила Галицького», Львів, Україна

Резюме. У статті висвітлено сучасну парадигму діагностики та активного ведення пацієнтів із харчовою алергією й анафілаксією у педіатричній практиці. Проаналізовано еволюцію клінічних критеріїв WAO та EAACI 2025 року, специфіку «масок» анафілаксії у дітей раннього віку та скасування жорстких часових рамок маніфестації. Наведено покроковий алгоритм невідкладної допомоги відповідно до найновіших рекомендацій ERC 2025 (Paediatric Life Support), принципи стратифікації ризику біфазних реакцій та перспективи використання назального адреналіну.

Мета дослідження проаналізувати два клінічні випадки пацієнтів з анафілаксією та пошук можливостей формування харчової толерантності, враховуючи сучасні алгоритми.

Матеріали та методи дослідження. Аналіз даних історій хвороби пацієнтів, результатів клінічних та лабораторних досліджень.

Результати дослідження. У дівчинки віком 4 роки було два випадки анафілаксії (11 місяців, 1 рік 3 місяці) після вживання льодяника з лізоцимом та після вживання яйця. Після тривалої елімінаційної дієти і проведених орально-провокаційних проб було розпочато лікування методом яєчної драбини. В динаміці состерігалося зменшення рівня специфічних антитіл до компонентів яйця: овомукоїд (Gal d 1) із 3,53 до 0,2 kU/L, овальбумін (Gal d 2) із 9,47 до 1,59 kU/L, лізоцим (Gal d 4) із 9,9 до 1,81 kU/L. Дитина успішно почала толерувати яйце з добрим рівнем термічної обробки. У хлопчика віком 2 роки 3 місяці зафіксовано два випадки анафілаксії (8 місяців, 1 рік 3 місяці) після вживання молочних продуктів. Результати проведеної молекулярної діагностики дозволили виявити високі рівні специфічних IgE до основних білкових компонентів коров'ячого молока: α-лактальбуміну (Bos d 4) — 37,45 kUA/L, β-лактоглобуліну (Bos d 5) – 28,25 kUA/L та казеїну (Bos d 6) — 32,45 kUA/L. З урахуванням анамнестичних даних, клінічної картини та результатів лабораторного дослідження рекомендовано повністю виключити молоко та всі продукти щонайменше на шість місяців.

Висновки. 1. Лікування пацієнта з харчовою анафілаксією в анамнезі, дозволяє шукати «вікно можливостей» для перегляду питання повторного введення продукту, але все це повинно бути підкріплене точною діагностикою, супроводом досвідченої команди в умовах стаціонару та виключно після періоду елімінації, яка має тривати щонайменше 6 місяців. 2. Пацієнти з високим ризиком анафілаксії мають бути забезпечені препаратом першої лінії – адреналіном і мати чіткі інструкції щодо дій.

Ключові слова: анафілаксія, діти, харчова алергія, молекулярна діагностика, адреналін.

Конфлікт інтересів. Автори заявляють про відсутність конфлікту інтересів.

Декларація етики. Під час підготовки клінічного випадку дотримано принципів конфіденційності персональних даних пацієнта. Інформацію подано в узагальненому вигляді без розкриття персональних ідентифікаційних даних.

Джерела фінансування. Дослідження не отримало зовнішньої фінансової підтримки.

Conflict of interest. The authors declare that there is are no conflicts of interest.

Ethics Declaration. Patient confidentiality principles were strictly observed accounted for during the preparation of this clinical case report. The information is presented in generalized form without disclosing any personal identification data.

Sources of Funding. This study did not receive any external financial support.

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Цитування: Беш ЛВ, Мацюра ОІ, Слюзар ЗЛ, Борисюк ОП. Анафілактичний шок у дітей, спричинений харчовими алергенами: сучасні підходи до діагностики та стандарти невідкладної допомоги в клінічних випадках. Астма та алергія. 2026;25(3):51–63. DOI: 10.31655/2307-3373-2026-25-3-51-63.

Cited: Besh LV, Matsyura OI, Slyuzar ZL, Borysiuk OP. Anaphylactic shock in children caused by food allergens: current approaches to diagnosis and standards of emergency care in clinical cases. Asthma and allergy (Ukraine). 2026;25(3):51–63. DOI: 10.31655/2307-3373-2026-25-3-51-63. Ukrainian.

Відомості про авторів

Л. В. Беш*
ДНТ «Львівський національний медичний університет імені Данила Галицького»
професор кафедри педіатрії та медичної генетики
Д. мед. н., професор
Вул. Пекарська 69, 79010, Львів, Україна,
E-mail: Lesya.besh@gmail.com
ORCID iD https://orcid.org/0000-0003-1897-7461

О. І. Мацюра
ДНТ «Львівський національний медичний університет імені Данила Галицького»
Завідувачка кафедри педіатрії та медичної генетики
Д. мед. н., професор
Вул. Пекарська 69, 79010, Львів, Україна,
E-mail: omatsyura @gmail.com
ORCID iD https://orcid.org/0000-0003-2656-259X

З. Л. Слюзар
ДНТ «Львівський національний медичний університет імені Данила Галицького»
Асистентка кафедри педіатрії та медичної генетики
Доктор філософії
Вул. Пекарська 69, 79010, Львів, Україна,
E-mail: zoryana.slyuzar @gmail.com
ORCID iD https://orcid.org/0000-0001-5001-8063

О. П. Борисюк
ДНТ «Львівський національний медичний університет імені Данила Галицького»
Доцентка кафедри педіатрії та медичної генетики
Вул. Пекарська 69, 79010, Львів, Україна,
E-mail: olenabora@gmail.com
ORCID iD https://orcid.org/0000-0002-4384-230X

Надійшла до редакції / Received: 01.07.2026 р.
Після доопрацювання / Revised: 24.08.2026 р.
Прийнято до друку / Accepted: 15.09.2026 р.

Information about autors

L. V. Besh*
State Nonprofit Company "Danylo Halytsky Lviv National Medical University"
Professor, Department of Pediatrics and Medical Genetics
Doctor of Medical Sciences, Professor
69 Pekarska St., 79010, Lviv, Ukraine,
E-mail: Lesya.besh@gmail.com

O. I. Matsyura
State Nonprofit Company "Danylo Halytsky Lviv National Medical University"
Head of the Department of Pediatrics and Medical Genetics
Doctor of Medical Sciences, Professor
69 Pekarska St., 79010, Lviv, Ukraine,
E-mail: omatsyura @gmail.com

Z. L. Slyuzar
State Nonprofit Company "Danylo Halytsky Lviv National Medical University"
Assistant of the Department of Pediatrics and Medical Genetics
PhD
69 Pekarska St., 79010, Lviv, Ukraine,
E-mail: zoryana.slyuzar @gmail.com

O. P. Borysiuk
State Nonprofit Company "Danylo Halytsky Lviv National Medical University"
Associate Professor of the Department of Pediatrics and Medical Genetics
69 Pekarska St., 79010, Lviv, Ukraine,
E-mail: olenabora@gmail.com