Food Allergy: How Labelling the Food you Serve can Save Lives

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ABSTRACT

Food allergy and associated anaphylaxis is an issue that plagues an unfortunate section of the world population and at times can prove fatal. Modern immunology has devised techniques for prevention, early diagnosis and to an extent, cure through desensitisation for food allergies. It is as important for clinicians to be aware of these developments to educate their patients, as it is for them to know how to manage a case of food-induced anaphylaxis in the emergency room. Labelling food items served at gatherings, with details of the ingredients and allergen information is a simple yet highly effective strategy to prevent fortuitous anaphylactic episodes and threats to life.

Keywords: Food Allergy, Anaphylaxis, Prevention, Treatment, Adrenaline, Food Hypersensitivity

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INTRODUCTION

Food allergy is defined as an adverse reaction to food in which immunological mechanisms have been demonstrated. It can result in considerable morbidity and has a detrimental impact on the quality of life of the individual. . It may cause life-threatening anaphylaxis and increase the cost of medical care.[1] This article focuses on food allergy as a cause of anaphylaxis and provides the latest consensus on the management of anaphylaxis.

BRIEF CASE REPORT

A 51-year-old male was brought to the Emergency Department (ED) of Government Medical College, Kozhikode with a sudden onset of dyspnoea and loss of consciousness while taking lunch. His vital signs on presentation were as follows:

HR: 113/min BP: 207/124 mmHg SpO2:49% on room air GRBS: 218 mg% GCS: E3V1M5

He was triaged to the Priority 1 area and started on oxygen. On chest auscultation, bilateral wheeze was present. On exposure, no rashes were found and the capillary refill time was less than 2 seconds. The bystander who accompanied the patient provided the history that the patient had told him just before collapsing that he had a shellfish allergy. The patient had also conveyed to the bystander that he might have eaten a dish containing shellfish at lunch and that the symptoms he was experiencing were probably due to it. After administering 0.5 mg Adrenaline IM stat and securing IV access, the patient was electively intubated from the ED with a presumptive diagnosis of anaphylaxis and shifted to the ED-ICU for intensive care.

The patient did not develop anaphylactic shock and could be extubated the next day and was later discharged home after full recovery. Upon enquiring about the history of events from the patient on the second day after extubating, he revealed that he had a dish at lunch on the day made as a thick paste of many different ingredients, and he could not identify what he was eating from its taste. Later after he started feeling a bit uneasy, he overheard two women talking

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about the said dish. They were saying that it was made from prawn meat. He quickly realised the symptoms as anaphylaxis and told his friend to take him to the ED before collapsing. The patient was a hospital staff and the friend was a healthcare worker trained in basic life support. Unfortunately, the food items and their ingredients were not labelled in this incident. The patient had a history of hypertension and bronchial asthma and was on medication for the same. The patient discovered his hypersensitivity towards shellfish while he was abroad 20 years ago, when he had an episode of life-threatening angioedema after eating shellfish at a restaurant. Timely recognition and management saved his life then.

DISCUSSION

Background

The World Allergy Organisation – Anaphylaxis Committee in 2020 defined anaphylaxis as follows:

"Anaphylaxis is a serious systemic hypersensitivity reaction that is usually rapid in onset and may cause death. Severe anaphylaxis is characterized by potentially life-threatening compromise in airway, breathing and/or the circulation, and may occur without typical skin features or circulatory shock being present."[2]

In 1902, Charles Robert Richet and Paul Portier (both French physiologists) were conducting experiments on dogs during an expedition organised by the then prince of Monaco, Albert I. They were trying to protect dogs from the toxins of a sea anemone called the Mediterranean Snakelocks.[3] After sensitising the dogs with the toxin, they found that repeat vaccinations with the toxin did not provide any protection. On the contrary, it produced vomiting, wheezing and death. They decided to coin the term anaphylaxis to label this lack of protection (ana = absence + phylaxis = protection in Greek). Charles Richet was later awarded the Nobel Prize in physiology/medicine in 1913 for this discovery.

Table 1. Major food allergens. Those marked by an asterisk are more common in children. Adapted from[2,10,11]

1	Milk*
2	Eggs*
3	Fish
4	Shellfish
5	Tree-nuts
6	Wheat
7	Peanuts*
8	Soybeans
9	Sesame

Epidemiology

Food allergy affects 2 – 4% of the population worldwide.[3] The global incidence of anaphylaxis is between 50 – 112 episodes per 100,000 person-years. The lifetime prevalence is 0.3-5.1%, varying according to different definitions used.[2] 1 in 300 people will experience anaphylaxis at some point in their lifetime.[5]

Triggers

Food allergens are the leading cause of anaphylaxis followed by insect venom and medications, in that

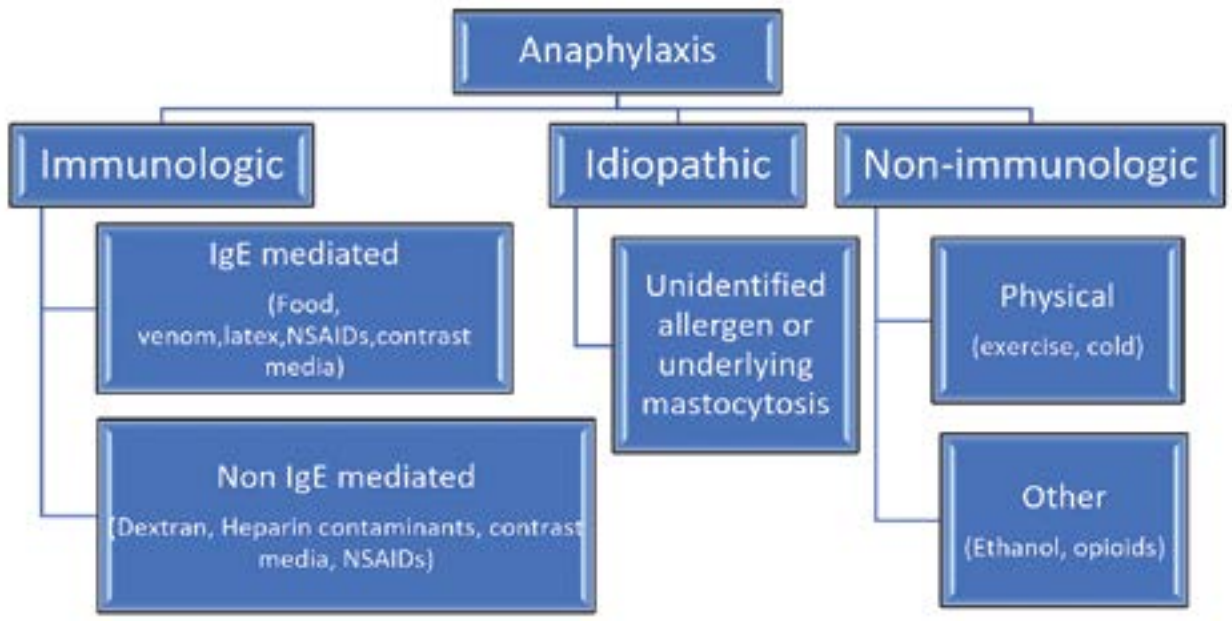

Figure 1. **Pathophysiologic classification of anaphylaxis**

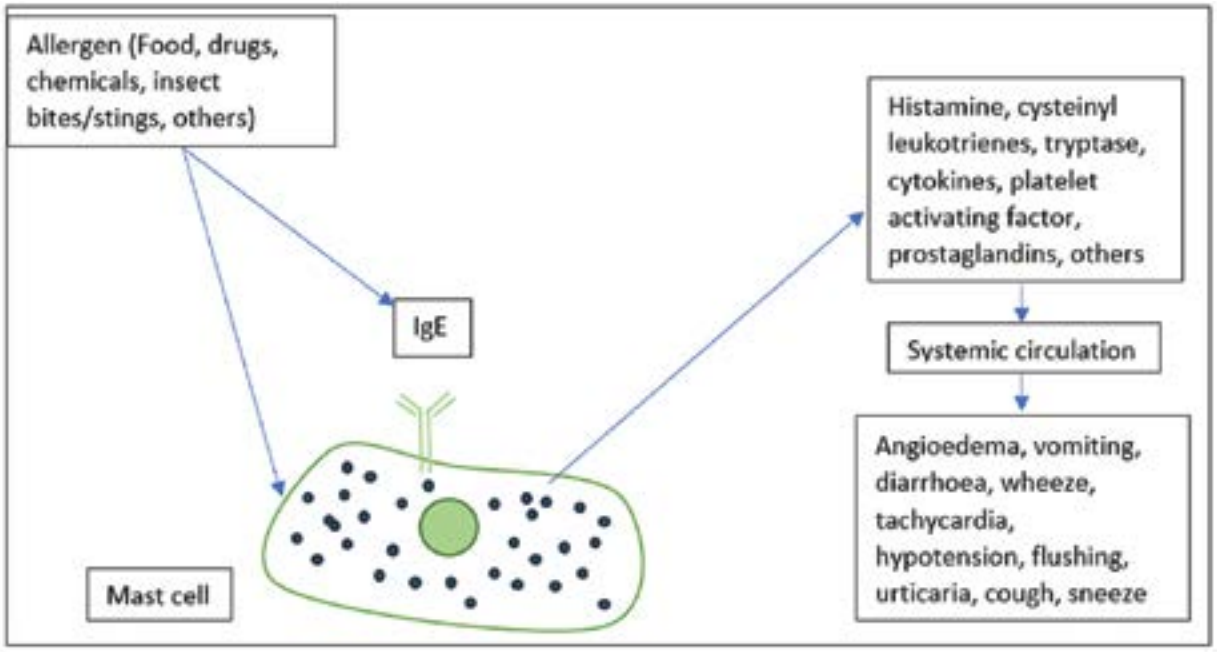

Figure 2. **Pathophysiologic mechanism of anaphylaxis**

order.[3,6] Drugs are the most common cause of fatal anaphylaxis and peanuts & other tree-nuts are the most common cause of food-induced fatal anaphylaxis.[7] Fish allergy contributes to 9% of fatal anaphylaxis. It persists from childhood to adulthood and is usually seen in coastal countries rather than landlocked countries.[8,9]

The 9 major food allergens are provided in **Table 1.**

A new type of food allergy was also discovered in the last decade, known as the alpha-Gal syndrome.[12] It has 2 modes of presentation – either as a delayed reaction, 2-6 hours after eating mammalian meat or immediately after receiving Cetuximab, a chemotherapy agent. It is caused by an IgE antibody response to galactose-alpha 1,3 -galactose (alpha Gal). This oligosaccharide is present in the saliva of some ticks, mammalian meat and in Cetuximab. Those who have been bitten by the tick, get sensitised to this antigen and later when they consume mammalian meat or receive Cetuximab, develop an anaphylactic reaction. The reaction is delayed in case of mammalian meat ingestion because of the time required for digestion of the meat and presentation of the antigen to the mast cells of the peripheral tissues.[12]

Pathophysiology

Anaphylaxis is a multiorgan phenomenon and a lot of cells contribute to its pathophysiology including mast cells, basophils, neutrophils, mast cells and platelets. It can be classified into idiopathic, immunologic and non-

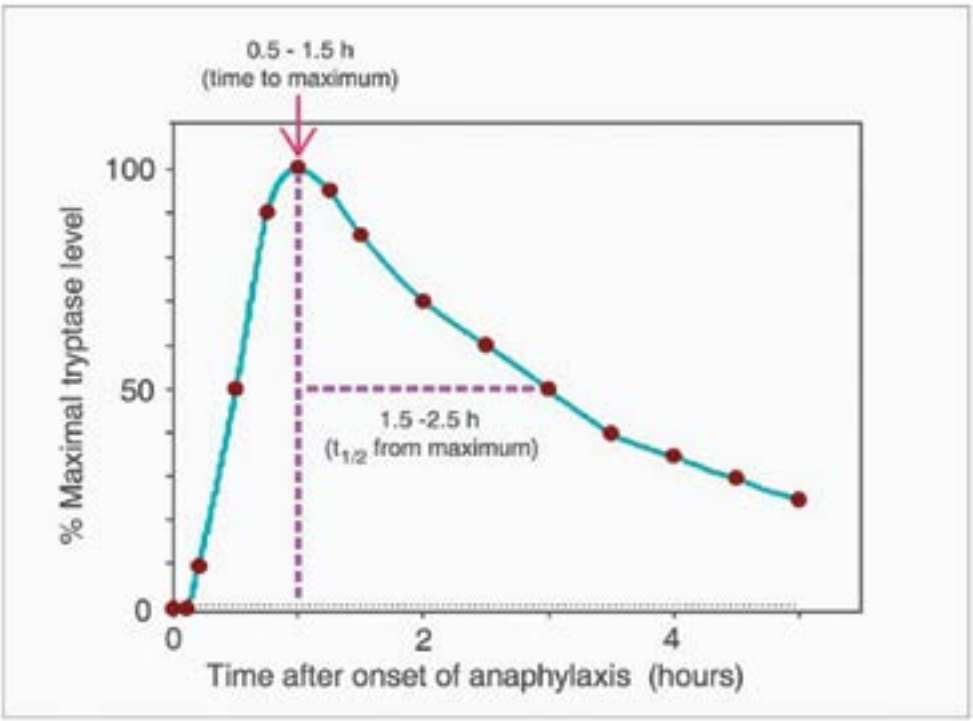

Figure 3. **Suggested time course for the appearance of tryptase in serum during anaphylaxis. Adapted from 'Emergency Treatment of Anaphylaxis May 2021' – Resuscitation Council UK 2021 guidelines.**

immunologic **(Figure 1).** Non-IgE mediated immunologic mechanisms include IgG-mediated anaphylaxis and complement activation. Radiocontrast media and certain drugs like NSAIDs have been shown to trigger anaphylaxis by more than one mechanism.[2]

Mast cells and basophils degranulate and release mediators that produce the systemic manifestations of anaphylaxis **(Figure 2).**

These mediators include histamine, cysteinyl leukotrienes, anaphylotoxins and platelet activating factor. They trigger symptoms like sneezing, angioedema, cough, wheeze, vomiting, diarrhoea, tachycardia, hypotension, flushing and urticaria. Anaphylaxis can produce changes in mediators like cytokines, prostaglandins and tryptase. The latter is a serine protease produced by mast cells and basophils which produces tissue oedema.

Tryptase levels in the serum

It can act as a biomarker as it increases to significant levels by around 30 minutes after symptom onset and reaches peak levels by 1-2 hours.[13] Tryptase levels > 12.4 ng/mL in the ED has a positive predictive value of up to 93%. The half-life of tryptase is short and its levels will reach back to baseline within 6-8 hours of the onset of the reaction. Hence the timing of the samples is very important.[13] See **Figure 3.**

Endogenous and exogenous cofactors

The severity of the anaphylactic reaction depends not only on the trigger and the mediators but also on certain cofactors[2,8,14] **(Table 2).**

Table 2. Cofactors of anaphylaxis

Endogenous cofactors	Exogenous cofactors
Underlying disease states (asthma, eczema, mastocytosis, cardiovascular disease) Coexisting allergy to other triggers, ongoing infection, hormonal status (pre-menstrual) Age-related factors (infants, adolescents, elderly).	Medication use (beta-blockers, ACE-inhibitors, NSAIDs, sedatives, anti-depressants) Ethanol and recreational drug use Psychological stress Physical exercise Disruption of routine

DIAGNOSIS

The World Allergy Organisation – Anaphylaxis Committee proposed the following criteria for diagnosis of anaphylaxis **(Table 3)**.

Table 3. Anaphylaxis is highly likely when any one of the following 2 criteria are fulfilled

1. Acute onset of an illness (minutes to several hours) with simultaneous involvement of the skin, mucosal tissue, or both (e.g. generalized hives, pruritus or flushing, swollen lips-tongue-uvula)
• AND AT LEAST ONE OF THE FOLLOWING
a) Respiratory compromise (e.g., dyspnoea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
b) Reduced BP or associated symptoms of end-organ dysfunction (eg, hypotonia [collapse], syncope, incontinence)
c) Severe gastrointestinal symptoms (e.g., severe crampy abdominal pain, repetitive vomiting), especially after exposure to non-food allergens
2. Acute onset of hypotension or bronchospasm or laryngeal involvement after exposure to a known or highly probable allergen for that patient (minutes to several hours), even in the absence of typical skin involvement.

- Hypotension is defined as a decrease in systolic BP greater than 30% from that person's baseline OR
  - i. Infants and children under 10 years: systolic BP less than (70 mm Hg + [2 x age in years])
  - ii. Adults and children over 10 years: systolic BP < 90 mm Hg
- Bronchospasm excludes lower respiratory symptoms triggered by common inhalant allergens or food allergens perceived to cause "inhalational" reactions in the absence of ingestion.
- Laryngeal involvement includes stridor, vocal changes and odynophagia.

Mast cell tryptase

Three timed samples of serum tryptase are to be obtained whenever the diagnosis is uncertain.

1. As soon as possible after symptom onset (without delaying treatment)
2. 1-2 hours after symptom onset (no later than 4 hours).
3. At least 24 hours after complete symptom resolution (to obtain baseline value).

   There are 3 definitions for a raised tryptase level.

   i. Values > 11.4 or 14 ng/mL (95 or 99% of the upper limit of normal)

   ii. Values > [(1.2 x convalescent sample tryptase) + 2] ng/mL

   iii. Any increase from the pre-reaction level. (pre-reaction tryptase level usually not available)

◊ ***A normal tryptase level does not rule out anaphylaxis***

Grade 1	Grade 2	Grade 3	Grade 4	Grade 5
Mild symptom/sign(s) only. NB: Reactions can be further categorized as: • **1t**: transient (<20 mins) • **1s**: single organ system only, ≥20 mins • **1m**: 2+ organ systems, ≥20 mins	Any 1 (or more) of the following moderate symptom/sign(s):	Any 1 (or more) of the following symptoms/signs:	Any 1 (or more) of the following symptom/ signs:	Any 1 (or more) of the following symptom/ signs:
Cutaneous (any one of): • Limited (few) or localized hives/urticaria* • Skin flushing (low areas of faint erythema) or mild pruritus* • Swelling (e.g. lip edema)* *excluding localized symptoms at application site	**Cutaneous** (any one of): • Systemic urticaria (e.g. numerous or widespread hives) • Generalized (>50% BSA) erythema • Widespread pruritus with protracted scratching • Significant angioedema (excluding lip swelling and laryngeal edema)	**Lower respiratory** • Bronchospasm (e.g. wheezing, shortness of breath) which responds to first line treatment • Cough due to laryngeal or lower respiratory involvement	**Respiratory** • Severe bronchospasm (not improving with 2 doses of IM epinephrine ± other appropriate treatment) • Stridor (with increased work of breathing)	**Respirator** • Respiratory failure requiring positive pressure ventilation • Respiratory arrest
Or	And/or	And/or	And/or	And/or
Upper respiratory • Nasal symptoms (e.g., sneezing, rhinorrhea, itch, congestion) • Throat-clearing (itchy throat)* or throat tightness/discomfort • Cough due to throat irritation or nasal symptoms		**Upper respiratory/laryngeal** • Throat tightness with vocal hoarseness • Stridor without increased work of breathing • Persisting (≥20 mins) odynophagia (pain on swallowing)	**Cardiovascular** • Hypotension with associated symptoms of end-organ dysfunction (e.g. hypotonia, dizzy[†], collapse[†], syncope) OR • decrease in systolic blood pressure (sysBP) ≥30% from that person's baseline OR • SysBP <90 mmHg in adults (in children ≤10 years, sysBP <70 mmHg + [2 × age in years]) †excluding vasovagal events (these present with dizziness/fainting which rapidly resolve on lying flat)	**Cardiovascular** • Anaphylactic shock i.e. requirement for IV vasopressor infusion to maintain sysBP ≥90 mmHg or MAP ≥65 mmHg in adults and children >10 years (or age-appropriate sysBP in younger children) • Cardiac arrest
Or		And/or		
Gastrointestinal • Nausea • Mild abdominal pain (for example, without a change in activity level)	**Gastrointestinal** • Persisting (≥30 mins) and non-distractable abdominal pain and/or • Vomiting (not due to gag or taste aversion) and/or diarrhea	**Gastrointestinal AND Cutaneous** Severe GI symptoms together with cutaneous features which meet WAO 2020 criteria for anaphylaxis (e.g. severe crampy abdominal pain, repetitive vomiting, especially after exposure to a non-ingested allergen)		
Or		And/or	And/or	
Other • Conjunctival reddening (not due to eye rubbing), pruritus, or tearing • Metallic taste		**Uterine** cramps + uterine bleeding	**Neurological** • Glasgow Coma Scale < 13	

Figure 4. **Adapted from[14] Turner et al. World Allergy Organization Journal (2024) 17:100876**

◊ *Tryptase levels also increase in severe asthma*

Diagnosis of food allergy is done by tests like the oral food challenge test, skin prick test, specific IgE test and elimination diets.[1] Since these tests cannot be done in the emergency room setting, these are not discussed further.

Grading of severity

In 2024, the World Allergy Organisation updated its grading system for systemic allergic reactions which is given in **Figure 4.**

MANAGEMENT

Anaphylaxis is a medical emergency requiring rapid treatment for survival. The steps to be followed once a provisional diagnosis of anaphylaxis is made, are as follows.

1. Remove exposure to trigger if possible. (e.g. discontinuing an IV agent or removing a sting)
2. Keep the patient in the supine position (In respiratory distress a sitting position will be better). If pregnant, position the patient in a semi-recumbent position on her left side. The benefit of elevating the lower limbs (Trendelenburg position) is controversial[2] In the pre-hospital setting, if the patient becomes unconscious, but is breathing normally, place the patient in the recovery position as shown in **Figure 5 (For more information, please see Appendix 1).**

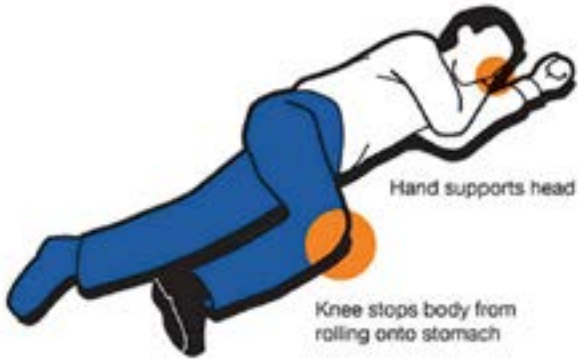

Figure 5. **Recovery Position in First Aid**
Source: Furst J. What is the Recovery Position in First Aid? [Internet]. 2023 [cited 2024 Jun 21]. Available from: https://www.firstaidforfree.com/what-is-the-recovery-position-in-first-aid/

Table 4. Dose of IM Adrenaline

Age	Intramuscular adrenaline dose
≥ 12 years	0.5 mg
6 – 11 years	0.3 mg
6 months – 5 years	0.15 mg
Infants < 6 months	0.10 – 0.15 mg

Table 5. Dose of oral cetrizine

Age	Dose of oral cetirizine
< 2 years	0.25 mg/kg
2 – 5 years	2.5 – 5 mg
6 – 11 years	5 – 10 mg
≥ 12 years	10- 20 mg

Table 6. Adrenaline availability and dosage

Adrenaline		
Availability	As 1 mL ampoules containing 1:1000 solution. Each 1 mL of 1:1000 solution contains 1 mg of adrenaline	
Dose	IM	0.5 mL of 1:1000 solution (0.5 mg) in anterolateral thigh
	IV infusion	Mix 1 mL of 1:1000 solution in 100 mL of saline and start at 0.5 – 1.0 mL/kg/hr

3. Administer intramuscular adrenaline stat in the vastus lateralis muscle (anterolateral thigh). The recommended dose is 0.5 mg (0.5 mL of 1:1000 solution) in adults and 0.01mg/kg in children < 12 years. Repeat every 5-15 minutes if symptoms do not resolve. A simplified age-based dosing of intramuscular adrenaline is given in **Table 4, 6.** In patients taking beta-blockers without optimal response to adrenaline, parenteral glucagon can be considered.[2]

4. **Conduct the primary survey:**

Airway: Airway adjuncts can be used or the patient intubated as necessary. Nebulised epinephrine can be considered in upper airway obstruction. The recommended dose is 5 mL of 1:1000 adrenaline.

Breathing: Start 100% oxygen via a non-rebreather mask for all patients in respiratory distress or requiring more than one dose of IM adrenaline. Nebulised short-acting beta-2 agonists can be given.

Circulation: Obtain IV access with a large bore cannula (14G or 16G in adults). Give crystalloid bolus of 20mL/kg in hemodynamically unstable patients.

Disability: Check the GCS (Glasgow Coma Scale) of the patient, pupils for size and reaction and blood glucose.

Exposure: Look for skin/mucosal changes. These may be patchy erythema, urticaria or angioedema. Skin/mucosal changes may be subtle/absent in 20% of reactions.[5]

5. **Antihistamines:** Antihistamines are of no benefit in treating the life-threatening features of anaphylaxis and their use may delay the administration of subsequent doses of adrenaline. Antihistamines can be used to treat cutaneous symptoms without delaying the treatment of respiratory and cardiovascular symptoms of anaphylaxis.[13] Once the patient is stable, an oral non-sedating antihistamine can be given (e.g. cetirizine) **(Table 5).**

   If the oral route is not possible chlorpheniramine can be given by IM or slow IV injection. There is no evidence to support the use of H-2 antihistamines.[13]

6. **Corticosteroids:** Evidence suggests that corticosteroids have no role in the acute management of anaphylaxis and hence are not recommended for the same.[2,13] However, they can prevent protracted symptoms in asthmatics as well as biphasic reactions. So, they can be given after the initial resuscitation and stabilisation. Early steroids are indicated in the case of asthmatics where the acute exacerbation of the asthma may have contributed to the severity of anaphylaxis.[13]

7. Refractory Anaphylaxis

It is defined as anaphylaxis requiring ongoing treatment (due to persisting respiratory or cardiovascular symptoms) despite two appropriate doses of IM adrenaline.[13] This accounts for < 1% of all anaphylactic reactions.

Treatment

i. **Fluid therapy:** Give IV fluid boluses. Use 500 mL boluses of crystalloids in adults and 10 mL/kg boluses of crystalloids in children < 12 years, till hypotension is corrected.

ii. **Adrenaline infusion:** The dose of adrenaline infusion is 0.05 – 0.5 ug/kg/min. This can be started by mixing 1mL of 1:1000 solution of adrenaline in 100mL of normal saline and infusing at 0.5 – 1.0 mL/kg/hour. Titrate according to response. Do not piggyback this line on another infusion line and do not infuse on the side of the BP cuff. Once symptoms start to improve, the infusion rate can be reduced. After all the symptoms have resolved, the infusion can be slowed progressively and stopped over 30 minutes. Monitor and observe for recurrence and restart infusion if necessary.

If refractory to adrenaline infusion consider adding a second vasopressor which may be noradrenaline or vasopressin and in patients on beta-blockers consider glucagon.

8) **Biphasic reaction:** A recurrence of symptoms several hours after onset in the absence of further allergen exposure is termed a biphasic reaction. It is difficult to distinguish from sustained anaphylaxis after a transient response to adrenaline or due to further absorption of allergen from residual food in the GI tract. This occurs in around 5% of patients and the median time of onset is 12 hours after initial symptom onset.

Risk factors for a biphasic reaction are

i. Severe initial presentation

ii. Delay in administering adrenaline (>30-60 min)

iii. Initial reaction requiring more than one dose of adrenaline.

iv. Previous history of biphasic reactions

Extended periods of observation of up to 12 hours are recommended for those patients with resolved symptoms, who developed severe reactions (e.g. hypotension or severe respiratory compromise) or required more than one dose of adrenaline.[3,13]

Learning point: Anaphylaxis due to C1 esterase inhibitor deficiency is resistant to adrenaline, steroids and antihistamines and needs treatment with C1 esterase inhibitor concentrate or fresh frozen plasma.

SUMMARY OF MANAGEMENT

Table 7. Summary of anaphylaxis management

Step no.	Measures to be taken
1	Remove Trigger
2	Position the patient
3	Administer IM adrenaline (0.5 mg IM i.e. 0.5 mL of 1:1000 solution)
4	Conduct primary survey
5	Adrenaline infusion – for refractory anaphylaxis or hypotension refractory to fluids (mix 1 mL of 1:1000 adrenaline in 100mL saline and infuse at 0.5 – 1.0 mL/kg/hr)
6	Antihistamines and steroids once patient is stabilised (Diphenhydramine 1mg/kg [max 50 mg] IV, Methylprednisolone 125-250mg [1-2 mg/kg in children] IV)
7	Monitor for biphasic reaction

PATIENT EDUCATION

Patients need to be educated on the following points.

- The reasons for anaphylaxis and the risk of recurrence
- Use Medical alert bracelets for immediate attention in situations of sudden collapse
- To carry an anaphylaxis emergency action plan with them to instruct rescuers on actions to be taken and how to use the epinephrine auto-injectors (EAI).
- Prescribe at least 2 epinephrine auto-injectors (EAI). EAIs must be prescribed to all patients who have had an anaphylactic episode except for those who had it in response to a medication unless it is not possible to avoid that medication in the future[13,15]
- The patient and family members must be taught how to use the EAI. It is to be injected in the anterolateral thigh and must be kept in place for at least 3 – 10 seconds.

PREVENTION OF FOOD ALLERGY

Primary prevention: The goal is to stop the onset of IgE sensitisation to a food-specific allergen. It targets the perinatal and infancy period. Having a balanced diet during pregnancy decreased the risk of food allergy in children, rather than the previous misconception of eliminating food allergens. The current emphasis is on a balanced diet for lactating mothers for nutrient-rich breast milk.[16] A systematic review and meta-analysis conducted in 2023 focussing on the introduction timing of multiple allergenic foods (milk, eggs, fish, shellfish, tree nuts, wheat, peanuts and soya) and their association with allergy prevention revealed that earlier introduction of multiple allergenic foods during infancy itself was associated with reduced IgE-mediated allergy to any food.[16]

Secondary prevention: In Oral immunotherapy (OIT) increasing doses of a food allergen (usually in a food vehicle) is administered to an allergic patient to increase the threshold at which they react to it. The EACCI currently recommends OIT for allergy to milk, eggs, peanuts and other tree-nuts.

Tertiary prevention: This includes avoiding exposure to the allergen and managing symptoms using epinephrine auto-injectors. It can reduce mortality, but not the prevalence of food allergy.

FOOD LABELLING

Simple measures can go a long way in preventing unwanted casualties.

1. Labelling food items is a highly effective means to prevent such events and save lives.
2. The use of precautionary allergen labelling (PAL) on packaged food products.[17] The Food Safety and standards (labelling and display) Regulations 2020 released by the FSSAI (Food Safety and Standards Authority of India) require Indian food producers to label the allergens contained in the food including those that may be present due to cross-contamination. This guideline mentions 8 classes of allergens.[18]
3. Ensure that whenever food is served at a public gathering, the major ingredients or atleast the common allergens contained in it are displayed.
4. Ask for previous h/o allergy to take extra care.
5. During preparation and serving, use extra caution to avoid preparing and serving different food items using shared utensils.

RECENT ADVANCES

1. Trans epidermal water loss (TEWL) monitoring: It can predict the onset of anaphylaxis before it is clinically evident. It is a non-invasive technique, where a device is kept on the skin of the volar forearm and an oral food challenge is given. TEWL increases during food allergy reactions and the magnitude of the rise was found to correlate with the rise in tryptase levels.[19]
2. Biological agents as an adjuvant to OIT: IgE plays an important in role in the pathophysiology of food allergy. Omalizumab is an anti-IgE monoclonal antibody already approved by the FDA for uncontrolled urticaria and asthma. It prevents IgE from binding to its receptors on mast cells, basophils, eosinophils and dendritic cells.[20]
3. Fish Allergen Immunotherapy: Of the studies that have been done, the majority have used cooked fish or fish extract for immunotherapy. Recently 3 studies were completed using fish allergens for immunotherapy (2 of them used parvalbumin, the major fish allergen).[9]
4. Nanoparticle therapeutics: They have been found to suppress the major allergic effector cells and disrupt the disease-producing immune pathways to produce more tolerogenic immune pathways. Some formulations that have shown efficacy are polymeric, lipid and emulsion-based-nanotherapeutics.[21]

CONCLUSION

Food-induced anaphylaxis is a preventable cause of death. Yet it continues to take lives in the modern world. Proper patient education, food labelling and awareness among those who serve food are all essential to prevent such episodes. Early recognition, intervention and management can treat the cases that occur despite these measures.

END NOTE

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Conflict of Interest: None declared

Appendix 1. The Recovery Position

The resuscitation council of the UK recommends the following steps to place a person (adult or child) who does not meet the criteria for initiation of rescue breaths or CPR in the recovery position.

- Kneel beside the person and make sure that both legs are straight.
- Place the arm nearest to you out at right angles to the body with the hand palm uppermost.
- Bring the far arm across the chest, and hold the back of the hand against the person's cheek nearest to you.
- With your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground.
- Keeping the hand pressed against the cheek, pull on the far leg to roll the person towards you onto their side.
- Adjust the upper leg so that both the hip and knee are bent at right angles.
- Tilt the head back to make sure the airway remains open.
- Adjust the hand under the cheek if necessary, to keep the head tilted and facing downwards to allow liquid material to drain from the mouth.
- Check regularly for normal breathing.
- Only leave the person unattended if absolutely necessary, for example to attend to other people.

Source: Adult basic life support guidelines from the 2021 resuscitation guidelines, Resuscitation Council UK.

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