Managing anaphylaxis in the office setting

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ABSTRACT

Background: *Although the definition of anaphylaxis for clinical use may vary by professional health care organizations and individuals, the definition consistently includes the concepts of a serious, generalized or systemic, allergic or hypersensitivity reaction that can be life-threatening or even fatal.*

Methods: *In this review, we presented the important topics in the treatment of anaphylaxis in the office setting. This review will discuss triggers and risk factors, clinical diagnosis, and management of anaphylaxis in the office setting.*

Results: *Anaphylaxis in the office setting is a medical emergency. It, therefore, is important to prepare for it, to have a posted, written anaphylaxis emergency protocol, and to rehearse the plan regularly. In this review, we presented the important steps in managing anaphylaxis in the office. Treatment of anaphylaxis should start with epinephrine administered intramuscularly at the first sign of anaphylaxis. Oxygen and intravenous fluids may be needed for moderate-to-severe anaphylaxis or anaphylaxis that is quickly developing or if the patient is unresponsive to the first injection of epinephrine. Antihistamine therapy is considered adjunctive to epinephrine, which mainly relieves itching and urticaria. Corticosteroids, with an onset of action of 4–6 hours, have no immediate effect on anaphylaxis.*

Conclusion: *To prevent near-fatal and fatal reactions from anaphylaxis, the patient, the family, and the physician must remember to follow the necessary steps when treating anaphylaxis. In anaphylaxis, there is no absolute contraindication for epinephrine.*

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The prevalence of allergic diseases is increasing worldwide, attributed in part to increased exposure to environmental allergens and pollutants; nevertheless, these diseases remain underdiagnosed and undertreated. The most severe form of allergy, anaphylaxis, has a lifetime prevalence between 0.05% and 2%.[1] The widely disseminated World Allergy Organization Anaphylaxis Guidelines were initially published in 2011 and were updated in 2012 and 2013.[2–4] An International Consensus of Anaphylaxis published in 2014 compared and contrasted major anaphylaxis guidelines currently in use worldwide.[5] Definitions of anaphylaxis for clinical use by health care professionals have all included the concepts of a serious, generalized or systemic, allergic or hypersensitivity reaction that can be life-threatening or fatal.[5] The term "anaphylaxis" should also be used in preference to terms such as "anaphylactic shock," "allergic reaction," "acute allergic reaction," "systemic allergic reaction," "acute immunoglobulin E [IgE] mediated reaction," "anaphylactoid reaction," or "pseudoanaphylaxis."[2,6] Likewise "epinephrine" is often used interchangeably with "adrenalin" but, in this article, the World Allergy Organization preferred term, "epinephrine" was used.

The most common triggers for anaphylaxis are food, medications, and venom. Anaphylaxis occurred after subcutaneous allergen immunotherapy (SCIT) and vaccinations are responsible for <3% of all episodes of anaphylaxis in an office setting. They are likely to be the most common cause of acute anaphylaxis in the allergist's office where SCIT is being given.[7]

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Triggers

Different anaphylaxis triggers (elicitors, causes) predominate in different age groups. Among 24,443 adults (mostly nonatopic, 80% female), with a mean age of 42 years (range, 16–83 years) admitted to a tertiary health care facility, 516 (2%) were diagnosed with anaphylaxis. Drugs were by far the most common trigger (91% of cases).[8] In contrast, in two studies in adults with a mean (standard deviation) age of 51 ± 16.9 years[9] and 44.3 years (interquartile range, 32–58 years),[10] respectively, food triggers were as common as drug triggers, followed by venom triggers,[9,10] honey ingestion,[11] and the spotted tussock moth caterpillar, *Lophocampa maculate*.[12] Idiopathic anaphylaxis is also reported.[13] In children, the most common trigger is food, with up to 90% of anaphylaxis due to food, followed by insect venom, and idiopathic anaphylaxis.[14–20]

Foods as Triggers for Anaphylaxis

In the United States, the most frequent foods that cause anaphylaxis are peanuts, tree nuts, shellfish, fish, milk, and egg.[15–18] However, different geographic areas often have different foods responsible for most food-induced anaphylaxis, *e.g.,* milk and sesame in Israel and shellfish in Hong Kong.[19,20] Foods likely account for up to 40% of all episodes of anaphylaxis in children and adults in the outpatient setting.[18,21] Patients allergic to peanuts and tree nuts, those with multiple food reactions, a history of allergic reactions to a small amount of food, and previous food-induced anaphylaxis of any severity are at risk for fatal anaphylaxis.[22,23] Fatal reactions are most commonly reported in adolescents and young adults, possibly related to risk-taking activities in this age group.[22,24]

Accurate diagnosis of a food allergy, which has the potential for severe anaphylaxis and fatality on future ingestion, can be challenging, particularly when the original reaction was mild and limited to the skin. The degree of percutaneous and serum specific IgE reactivity correlate with the likelihood of being clinically allergic to the food but do not indicate the severity of the next reaction.[25] Food component–resolved diagnostic testing is becoming more widely available, and, for a few foods, there is research that a specific component(s) improves specificity and helps to identify those patients at higher risk.[25] For example, children who clinically reacted to peanut (including those with anaphylaxis) had higher specific IgE levels to Ara h 1, Ara h 2, and Ara h 3 than children who were asymptomatic peanut sensitized ($p < 0.00001$). Elevated specific IgE to Ara h 2 was the major contributor to accurate discrimination between clinical reactiv-

ity to peanut and asymptomatic sensitization to peanut (99.1% sensitivity, 98.3% specificity, and 1.2% misclassification rate) and had a higher discriminative accuracy than IgE to whole peanut extract ($p = 0.008$).[26] However, no current diagnostic test can accurately predict the severity of future reactions.[27]

Most infants and young children with anaphylaxis are atopic and often have comorbid allergic conditions[28] In a retrospective study of 371 infants, children, and teenagers with acute allergic reactions to food, the importance of underlying asthma was confirmed. During anaphylaxis, 72% of those with concomitant asthma had lower airway symptoms compared with only 49% of those without concomitant asthma ($p < 0.01$).[29] The significance of asthma as a risk factor is clearly demonstrated in a case study from Israel, in when four young patients died after ingesting small amounts of milk ($n = 3$) or hazelnut ($n = 1$) to which they had previously experienced allergic reactions.[30] Although all of these patients had concurrent asthma for which an inhaled bronchodilator had been prescribed, none were on a controller medication.

Food-induced anaphylaxis almost always requires ingestion of the food. In rare circumstances, mucosal contact, either direct or indirect (kissing a person who recently ingested the allergic food) can cause anaphylaxis. Although cooking fumes (*e.g.,* shellfish) have occasionally been reported to induce anaphylaxis, this is most unusual.[31] Reassuredly, there is no convincing evidence that skin contact or inhalation of food odors can induce near-fatal or fatal anaphylaxis, although local skin reactions after skin contact and wheezing after food inhalation have been reported.[31,32]

There are several recently described new forms of food-induced anaphylaxis. Oftentimes the food contains hidden allergens. Short-chain low-molecular-weight galacto-oligosaccharides with prebiotic effects that are added to some cow's milk formulas have been identified as a new trigger of IgE-mediated anaphylaxis in patients, median age of 6 years, with what seems to be milk allergy.[33] In tropical climates, orally ingested mites that contaminate wheat flour can trigger anaphylaxis even after cooking (the so-called pancake syndrome) and also play a role in food-dependent exercise-induced anaphylaxis.[34] A delayed onset of anaphylaxis of up to 6–8 hours has been reported in the United States, Australia, and Europe after ingestion of mammalian meats, *e.g.,* beef, port, and lamb. The relevant IgE reaction is to the carbohydrate moiety, galactose-α-1, 3-galactose, which seems to be related in may cases to previous sensitization to galactose-α-1, 3-galactose by having been bitten by ticks.[28]

Venoms as Triggers for Anaphylaxis

Anaphylaxis to stings affects up to 8% of the population worldwide and is likely underdiagnosed. Similarly, venom-induced fatality may be mistakenly attributed to heart attacks and strokes.[35,36] Unfortunately, ~50% of patients who die from venom stings did not know that they were allergic to venom.[36] If diagnostic test is positive to multiple stinging insects and there are true double positivity results to bee and vespid venoms, it is difficult to distinguish the cross-reactivity to these venoms.[37]

When reviewing postmortem serum of sudden deaths with cause unknown, many patients are found to have both elevated venom IgE and elevated tryptase levels.[38] It is recommended to obtain a serum tryptase level on all patients with venom allergy as well as those patients with idiopathic anaphylaxis because systemic mastocytosis or mast cell activation syndrome may also be present but undiagnosed.[39]

Drugs and Biologic Agents as Triggers for Anaphylaxis

Medications are the most common cause of anaphylaxis in adults. In a retrospective review, anaphylaxis comprised 6% of 16,157 adverse drug reactions and was reported in patients 7 days to 91 years old. Of these patients, 19% were hospitalized and 3% died.[40] The most common classes of drugs that cause anaphylaxis are (1) antibiotics, especially β-lactam antibiotics, and (2) nonsteroidal anti-inflammatory agents. Perianesthetic anaphylaxis is perhaps the third most common cause of anaphylaxis from medications.[41] In a 10-year audit of anaphylaxis to muscle relaxants, 20% of 220 patients had positive intradermal test results to the muscle relaxant given during their surgical procedure, most commonly rocuronium or suxamethonium; 65% of those who reacted to rocuronium and 29% of those who reacted to suxamethonium had cross-reactivity to another muscle relaxant.[42] More recently, antineoplastic and cytotoxic drugs, especially the platinum-containing drugs, biologic modifiers, and monoclonal antibodies have been responsible for anaphylaxis.[41]

Risk factors for overall drug allergy include (1) female sex; (2) a personal history of drug allergy; (3) recurrent drug exposure; (4) a long hospitalization time; (5) human leukocyte antigen type; and (6) certain disease states, *e.g.,* Epstein-Barr virus, systemic lupus erythematosus, and human immunodeficiency virus.[41,43–45] Atopy, age, and genetic differences in drug metabolism have not been demonstrated to be independent risk factors for drug allergy. Although female patients are at risk for overall drug reactions, this does not hold true for penicillin allergy. Proton pump inhibitor administration may increase the risk of developing any drug hypersensitivity. In 161 hospitalized patients, after controlling for confounders, the odds ratio of confirmed drug hypersensitivity was 4.35 (95% confidence interval, 2.0–9.45) in those who received a proton pump inhibitor compared with matched controls.[45]

Allergen Immunotherapy as a Trigger for Anaphylaxis

For allergy practices that administer SCIT, it is likely that this will be the number one trigger for office-based treatment of anaphylaxis. Although the incidence of anaphylaxis that involves a single system organ system ("grade 1" reactions of the World Allergy Organization SCIT grading system)[46] has been described in up to 7% of treated patients on conventional build-up regimens and up to 39% of accelerated dosing regimens, severe anaphylactic reactions are very uncommon.[46–49] Near-fatal reactions have been reported in the United States to occur at a rate of one in every million injections and fatal reactions at a rate of one in 2.5 million injections.[50,51] Although a few case reports have been published that describe anaphylaxis from sublingual immunotherapy, there are no reported fatalities.[52–55]

Strategies to prevent near-fatal and fatal reactions to SCIT include (1) avoiding, when possible, the administration of SCIT to patients on β-blockers; (2) using a preinjection questionnaire to review changes in the patient's medical condition, *e.g.,* episodes of asthma since the previous injection; (3) using standardized forms and procedures for SCIT; (4) using an objective measure of airway function (*e.g.,* peak flow measurement) for the patient with asthma before allergy injections; (5) insisting on a 30-minute waiting period after SCIT; and (6) giving consideration to prescribing a dual-pack epinephrine autoinjector to all patients on SCIT.[56]

Clinical Diagnosis of Anaphylaxis

The rapid diagnosis of anaphylaxis in an individual patient at a specific point in time can be very difficult because there are >40 signs and symptoms of anaphylaxis, some of which can be difficult to recognize.[57] The signs and symptoms present rapidly, after recent (minutes to a few hours) exposure to a known or presumed trigger, and usually involve more than one body system. Although skin manifestations, *e.g.,* urticaria and angioedema, are the most common presenting sign of anaphylaxis, other systems, *e.g.,* the gastrointestinal or cardiovascular system may be responsible for the first symptoms to appear.[41] (see Table 1 for signs and symptoms of anaphylaxis).[58–63] It is the responsibility of the health care provider to become familiar with all of the presenting patterns of anaphylaxis. A patient with diagnosed anaphylaxis must be educated on how to avoid his or her

Table 1 **Symptoms and signs of anaphylaxis***

	Symptoms and Signs of Anaphylaxis
Cutaneous, subcutaneous, mucosal tissue	Flushing, pruritus, hives (urticaria), swelling, morbilliform rash, pilor erection
	Periorbital pruritus, erythema and swelling, conjunctival erythema, tearing
	Pruritus and edema of the lips, tongue, uvula and/or palate
	Pruritus in the external auditory canals
	Pruritus of genitalia, palms, soles
Respiratory	Nose: pruritus, congestion, rhinorrhea, sneezing
	Upper airway obstruction from angioedema of the tongue, oropharynx, or larynx
	Larynx: pruritus and tightness in the throat, dysphonia and hoarseness, dry staccato cough, stridor, dysphagia
	Lung: shortness of breath, chest tightness, deep cough, wheezing and/or bronchospasm (decreased peak expiratory flow)
	Cyanosis
Gastrointestinal	Nausea, cramping abdominal pain, vomiting (stringy mucus), diarrhea
	Hyperperistalsis with fecal urgency or incontinence
Cardiovascular	Chest pain, palpitations, tachycardia, bradycardia, or other dysrhythmia
	Feeling faint, altered mental status, hypotension, loss of sphincter control, cardiac arrest; hypovolemic shock, syncope
Central nervous system	Aura of impending doom, uneasiness, throbbing headache, dizziness, confusion, tunnel vision; in infants and children, sudden behavioral changes, such as irritability, cessation of play, and clinging to parent
Other	Metallic taste in the mouth
	Dysphagia
	Uterine contractions in postpubertal female patients
	Urinary urgency or incontinence

**Adapted from Refs. 58–63.*

known triggers and how to rapidly recognize the signs and symptoms of early anaphylaxis.

Although anaphylaxis may be difficult to diagnose in a fully staffed and equipped office, when resources are limited, *e.g.,* when pulse oximeters, sphygmomanometers with appropriate arm-cuff sizes, and, at times, even electricity are unavailable, it indeed can be challenging.[64–68] In such situations, assessment of hypoxemia is based on clinical indicators, such as central cyanosis, nasal flaring, inability to speak or drink, grunting, lethargy, chest retractions, or a rapid respiratory rate. Assessment of hypotension and distributive shock is based on clinical indicators, such as weak or nonpalpable peripheral pulses.[64,68]

Management of Anaphylaxis

Anaphylaxis is a medical emergency. It, therefore, is important to prepare, to have a posted, written anaphylaxis emergency protocol and to rehearse the plan regularly. As soon as the clinical diagnosis is made, exposure to the trigger should be stopped if possible (*e.g.,* discontinuation of an intravenously (IV) administered diagnostic or therapeutic agent). The patient's circulation, airway, breathing, mental status, skin, and body weight (mass) should be rapidly assessed.[56] Simultaneously and promptly, a call for help should be made to a resuscitation team or to emergency medical services, if such support is available.[2,56]

Steps in the Management of Anaphylaxis in the Office Setting

The steps in the management of anaphylaxis in the office setting include the following[2,56]:

1. Develop an ongoing education program for all professional medical and clerical staff and for patients on how to recognize the signs and symptoms of anaphylaxis.
2. Have a written emergency protocol for recognition and treatment of anaphylaxis and rehearse it regularly.
3. Remove exposure to the trigger if possible, *e.g.,* discontinue an IV diagnostic or therapeutic agent that seems to be triggering the symptoms.
4. Assess the patient's circulation, airway, breathing, mental status, skin, and body weight.
5. Inject epinephrine (adrenaline) intramuscularly (IM) in the midanterolateral aspect of the thigh, 0.01 mg/kg of a 1:1000 (1 mg/mL) solution, maximum of 0.5 mg (adult) or 0.3 mg (child); record the time of the dose and repeat it in 5–15 minutes if needed. Most patients respond to 1 or 2 doses.
6. Call for help from resuscitation team (hospital) or emergency medical services (community) if available, for any patient not immediately responding to the first dose of epinephrine, any patient who requires IV fluids, for moderate-to-severe anaphylaxis, or whenever the patient becomes unstable or unresponsive to treatment.
7. Place the patient on his or her back, or in a position of comfort if there is respiratory distress and/or vomiting; place the pregnant patient on her left lateral side. Recognize that fatality can occur within seconds if the patient stands or sits suddenly.
8. When indicated, give high-flow supplemental oxygen (6–8 L/min), by facemask or oropharyngeal airway. Monitor oxygen saturation with pulse oximetry, if available.
9. Establish IV access by using needles or catheters with a wide-bore cannula (14–16 gauge). When indicated, give 0.9% (isotonic) saline solution rapidly (*e.g.,* 5–10 mL/kg in the first 5–10 minutes to an adult; 10 mL/kg to a child).
10. For persistent wheezing or stridor, administer inhaled β-agonists.
11. When indicated at any time, perform cardiopulmonary resuscitation with continuous chest compressions.
12. At frequent, regular intervals, monitor patient's blood pressure, cardiac rate and function, respiratory status, and oxygenation (monitor continuously, if possible).
13. Start cardiac monitoring as soon as equipment and skilled personnel are available.

14. Develop a personalized "Anaphylaxis Action Plan" for all patients diagnosed with anaphylaxis. Educate the patient on how to recognize the signs and symptoms of anaphylaxis, both an immediate and a biphasic reaction.

15. Prescribe epinephrine for self-injection, preferably an epinephrine autoinjector, and instruct the patient in proper administration technique. Each patient should have two doses of epinephrine, which should be available at all times. Advise the patient exactly when to administer epinephrine and how to access emergency assistance.

16. Observe the patient who is experiencing anaphylaxis in the office until stable. International guidelines do not always agree on an exact amount of minutes or hours this observation time should be. Observation times will also vary based on the severity of the reaction, if the patient required emergency department treatment, and the availability of follow-up care after discharge. If transportation by emergency medical services is required, then most guidelines recommend a minimum of 4 hours of observation. There should be a plan for patient follow-up by telephone within a few hours of discharge and then a planned office visit follow-up within a couple of weeks.

Epinephrine

The one drug that must always be administered first and fast is epinephrine (adrenaline). Epinephrine maintains blood pressure, opens airways, antagonizes the effects of the released mediators, and inhibits further release of mediators. Health care professionals are sometimes reluctant to administer epinephrine for fear of adverse effects. However, the use of epinephrine for anaphylaxis has no absolute contraindications. It is the drug of choice, and it is usually well tolerated and potentially lifesaving.[2,56,69–71] Anaphylactic deaths correlate with delayed administration of epinephrine. The initial dose can be repeated as necessary, depending on the response. Data are limited concerning the frequency with which patients might require repeated doses of epinephrine to treat anaphylaxis (reports range from 16 to 36%) and multiple cofactors might be involved.[2,56,69]

It is important to immediately administer epinephrine in the IM route.[72,73] The administration of epinephrine IM in the thigh (vastus lateralis) results in higher and more rapid maximum plasma concentrations of epinephrine than IM or subcutaneous administration in the arm (deltoid) of children and adults who are asymptomatic.[2] Obesity or other conditions that enlarge the subcutaneous fat pad may prevent adequate IM access.[2,56]

Oxygen

Oxygen is the second most important treatment of anaphylaxis and should be started for any patient with hypotension or any patient who is showing signs of respiratory distress or reduced oxygen saturation. If pulse oximetry is available, then administer oxygen for all patients with an oxygen saturation of <95%. Oxygen should be started for any patient who requires more than one injection of epinephrine. It should also be used for any patient with preexisting hypoxemia or myocardial dysfunction.[56]

Management of Blood Pressure

Because hypotension in anaphylaxis is due to a dramatic shift of intravascular volume, the fundamental treatment intervention after epinephrine is aggressive IV fluid administration. Large volumes of crystalloid, *e.g.,* 0.9 NaCl (isotonic) administered at 5–10 mg/kg within the first 10 minutes and continued to achieve 20–30 mg/kg within the first hour may be required.[56] The total volume may potentially exceed 5 L. The exact amount should be individualized and based on blood pressure and urine output. Refractory hypotension, depending on its severity, may require placement of an invasive cardiovascular monitor (central venous catheter) and arterial line once advanced support is available. Vasopressors may also be needed to support blood pressure. IV epinephrine (1:10,000 v/v preparation) can be administered as a continuous infusion, especially when the response to IM epinephrine (1:1000 v/v) is poor. Dopamine infusion can also be used. Without the availability of an infusion pump and cardiac monitor, these medications should be used very cautiously because they introduce increased cardiovascular risk to the patient.

Preexisting Patients with Heart Disease

In patients with or without preexisting heart disease, ischemic myocardial dysfunction may occur due to hypotension and/or hypoxia. Epinephrine, nonetheless, is required for all patients with severe anaphylaxis, regardless of the underlying cardiac status. If cardiac ischemia occurs due either to the effects of anaphylaxis on the heart or the combination of epinephrine with the underlying cardiovascular disease, then it can be treated once the anaphylaxis is stabilized. If pulmonary congestion or evidence of cardiac ischemia is present, then fluid resuscitation should be approached more cautiously once the anaphylaxis has responded to treatment.

Patients on β-Blockers

Patients with anaphylaxis who are taking a β-adrenergic blocking agent (*e.g.,* for hypertension, migraine prophylaxis) can have refractory anaphylaxis that is poorly responsive to standard measures. Data are limited to case reports, but glucagon, 1–5 mg IV over 5 minutes, may be effective in this situation.[56,74] Glucagon has both inotropic effects and chronotropic effects on the heart by increasing intracellular levels of cyclic adenosine 3,'5'-monophosphate, independent of the β-adrenergic receptors. Glucagon can also reverse bronchospasm.[41]

Antihistamines and Corticosteroids

The traditional treatment of anaphylaxis has always included antihistamines and corticosteroids, although the clinical benefit has been questioned. Until more research is available, most clinicians will administer antihistamines and corticosteroids as second-line therapy. However, antihistamines have a much slower onset of action than epinephrine and require at most 1 hour for peak activity, they exert minimal effect on blood pressure, and they should never be administered as the only treatment for anaphylaxis.[56,75]

Antihistamines, both H_1 (*e.g.,* diphenhydramine, cetirizine) and H_2 (*e.g.,* ranitidine) are considered second-line medications. When used for IV administration, diphenhydramine and ranitidine are appropriate agents. However, oral antihistamines may suffice for milder anaphylaxis. Some guidelines indicate that one should avoid all sedating oral antihistamines, *e.g.,* diphenhydramine, when possible, because this may result in sedation and further impair the proper assessment of the status of anaphylaxis. One should administer, instead, second-generation, less-sedating antihistamines, *e.g.,* cetirizine, which are available in the oral form.[6] Corticosteroids have no immediate effect on anaphylaxis because their onset of action is 4–6 hours after administration, regardless of the route of administration.[56,75]

Preparing the Office and Staff for Anaphylaxis

Education in anaphylaxis causes, signs and symptoms, and treatment must be a structured and ongoing process for both staff and patients. For SCIT and special procedures, *e.g.,* drug and food allergy challenges, it is suggested that all patients sign a consent form that details the risks and benefits of the procedure or treatment. All medical staff need to be current in cardiopulmonary resuscitation training. There should be a well-supplied and up-to-date "Anaphylaxis Emergency Cart," where all the emergency medications and supplies are kept together in one place. This cart should be easily moved to the patient who is experiencing anaphylaxis. In addition to the medications described above, pulmonary support and fluid access supplies should also be available.[41,56] The "Office Anaphylaxis Man-

agement Plan" that the office adopts should be posted throughout the office in easily accessible locations.[56]

CONCLUSION

All offices that administer SCIT, vaccines, and IV medications, and that conduct drug and/or allergy testing, oral challenges, and other at-risk procedures should have proper planning, preparation, and practice to respond to the early symptoms and signs of anaphylaxis. Staff and patient education, the "Anaphylaxis Emergency Cart," and the "Action Plan for Anaphylaxis Management" are all integral parts of the foundation for successful treatment of anaphylaxis. Treatment of anaphylaxis should start with epinephrine administered IM at the first sign of anaphylaxis. Oxygen and IV fluids may be needed for moderate-to-severe anaphylaxis or anaphylaxis that is quickly developing or unresponsive to the first injection of epinephrine. Emergency medical services should be called for all patients who are experiencing moderate-to-severe ("grade 2" reactions or higher) anaphylaxis,[46] if they require more than one dose of epinephrine and/or IV fluids, or if they do not immediately respond to treatment.[56] After a successful response to treatment, the patient must be observed until fully stable, educated in avoidance of allergic triggers, instructed on how to recognize the signs and symptoms of anaphylaxis, and is prescribed self-injectable epinephrine. Foremost, the patient must be advised exactly when to self-administer the epinephrine. In the event of anaphylaxis, there are no absolute contraindications for epinephrine.

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