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Emergency of outpatient anaphylactic shock: Review article

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***Abstract*---Background:** Anaphylaxis is a severe, potentially life-threatening allergic reaction triggered by substances such as food, medications, insect stings, or environmental factors. It presents a range of symptoms, including respiratory, cardiovascular, dermatological, and gastrointestinal manifestations, which can develop rapidly. Misdiagnosis is common, as symptoms overlap with conditions such as septic shock or asthma. The immediate administration of intramuscular epinephrine is critical for treatment, along with airway management, antihistamines, and glucocorticoids. **Aim:** This review aims to explore the outpatient management of anaphylactic shock, focusing on symptoms, treatment strategies, and emergency preparedness. The review emphasizes the importance of rapid intervention and staff training for successful management. **Methods:** Methods involve analyzing current literature on anaphylaxis, its clinical presentation, and emergency treatment protocols. **Results:** Results

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show that early epinephrine administration is crucial for improving outcomes, with delayed treatment contributing to increased fatality risks. Regular preparedness, such as maintaining an anaphylaxis cart and conducting staff drills, is vital for effective outpatient care. **Conclusion:** The conclusion underscores the necessity of equipping outpatient settings with proper protocols and emergency supplies to manage anaphylactic emergencies and the need for patient education on recognizing and managing future episodes.

***Keywords*---**anaphylaxis, outpatient emergency, epinephrine, airway management, immunoglobulin E, allergic reaction, shock, emergency preparedness.

Introduction

Anaphylaxis represents a severe and potentially fatal reaction, manifesting through respiratory, cardiovascular, dermatological, or gastrointestinal symptoms due to exposure to a triggering agent, typically a food, insect sting, medication, or environmental factor. Annually, it leads to approximately 1,500 fatalities in the United States [1,2]. In some instances, anaphylaxis may be misdiagnosed as septic shock, asthma, airway obstruction, panic attacks, or other conditions [3-5]. Diagnostic support can be provided through the measurement of urinary and serum histamine levels, plasma tryptase, and platelet-activating factor levels after symptom onset [3,6,7]. Immediate treatment is vital, with intramuscular epinephrine, proper patient positioning, and intravenous fluids being the primary interventions for acute care [4,8]. Additional treatments involve airway management, antihistamines, glucocorticoids, and beta-agonists [4]. Patients should be monitored for delayed or prolonged anaphylaxis and advised on initiating emergency treatments for future episodes [9,10]. A notable portion of the U.S. population remains at risk for anaphylaxis, with many cases being underrecognized and undertreated [2,11]. While anaphylaxis is driven by immunoglobulin E (IgE), anaphylactoid reactions occur without IgE mediation [4]. Due to their clinical overlap, this text uses "anaphylaxis" to describe both conditions. In line with the theme of this collection, the discussion centers on outpatient emergencies, specifically the symptoms, treatment, and management of anaphylaxis, with limited focus on diagnostic testing and differential diagnosis considerations.

Symptoms

Anaphylaxis can present with a variety of symptoms, with cutaneous manifestations such as urticaria and angioedema being the most frequent, occurring in 62% to 90% of cases [12]. However, the absence of skin symptoms does not rule out anaphylaxis, as severe cases characterized by rapid cardiovascular collapse and shock may occur without cutaneous signs [13]. Respiratory involvement is also common, with symptoms including dyspnea, wheezing, and upper airway obstruction due to edema [12]. Gastrointestinal symptoms such as nausea, vomiting, diarrhea, and abdominal pain, along with cardiovascular manifestations like dizziness, syncope, and hypotension, affect approximately one-third of patients [14]. Less frequent symptoms include

headache, rhinitis, substernal pain, pruritus, and seizures [12]. Anaphylaxis can also present in atypical ways, such as syncope without other accompanying symptoms [15].

The onset of symptoms can vary significantly, typically appearing within seconds to minutes following exposure to a trigger [2,5]. A newly discovered IgE antibody to a mammalian oligosaccharide has been linked to two distinct forms of anaphylaxis: an immediate reaction to cetuximab and a delayed reaction, occurring 3 to 6 hours after consuming mammalian products such as beef and pork [16,17]. This red meat allergy has been associated with tick bites and shows a growing prevalence in the southern and eastern regions of the United States [18]. Anaphylaxis may also be prolonged, lasting over 24 hours, or may recur after an initial resolution of symptoms [10].

In terms of clinical presentation, common signs and symptoms include urticaria, angioedema, upper airway edema, flushing, dyspnea, wheezing, dizziness, syncope, hypotension, nausea, vomiting, diarrhea, and cramping abdominal pain. Less common symptoms may include rhinitis, headache, chest pain, pruritus without rash, and palmar erythema with pruritus. For effective evaluation, it is important to consider the patient's history, including recent food, medication intake, physical activities, environmental exposures, and any associated tick or insect bites, as well as details about symptom onset, recurrence, and any prior similar episodes. Additionally, the impact of stress, recent illness, travel history, and the patient's perceived emotional state prior to the event may provide important diagnostic clues.

Treatment

Outpatient treatment for anaphylaxis requires proper planning, which includes educating both staff and patients, preparing an emergency anaphylaxis cart, and developing a comprehensive office action plan for managing anaphylaxis. At the onset of anaphylaxis, immediate actions should include administering intramuscular epinephrine in the mid-outer thigh, removing the inciting allergen (e.g., stopping an infusion), rapidly assessing airway, breathing, circulation, and mental status, and calling for assistance from staff members. Cardiopulmonary resuscitation (CPR) may be initiated if necessary, and emergency medical services (EMS) should be summoned [4]. After epinephrine is administered, EMS should be contacted for severe cases or if the patient does not respond to the initial epinephrine dose.

An outpatient emergency cart for anaphylaxis treatment should include essential items such as a stethoscope, sphygmomanometer, tourniquets, syringes, IV catheters, aqueous epinephrine or an autoinjector, airway support devices, portable oxygen, antihistamines (H1 and H2), corticosteroids, ammonia inhalants, intravenous fluids, and a glucagon kit for patients on beta-blockers. Other recommended items include an infusion pump and external defibrillator. Aside from medication administration, it is crucial to position patients supinely unless there is respiratory compromise, in which case alternative positioning may be required. Pregnant patients should be placed on their left side. After administering epinephrine, intravenous access should be established promptly to maintain hemodynamic stability, and supplemental oxygen may be provided to certain

patients. Rapid airway assessment and maintaining patency using the least invasive effective method (e.g., bag-valve-mask) should be prioritized [4,19]. For patients experiencing circulatory collapse or those unresponsive to intramuscular epinephrine, intravenous fluid replacement with normal saline is advised. A nebulized beta2-agonist (e.g., albuterol) may be administered for bronchospasm, and glucagon should be considered for patients on beta-blockers who do not respond to epinephrine [4,8,20,21]. H1 and H2 antihistamines or corticosteroids should not replace epinephrine as the primary treatment for anaphylaxis and are considered adjunctive therapies [8]. The observation period should be individualized, with extended observation of at least 4 to 8 hours for high-risk patients or those with a history of severe anaphylaxis [9,10]. It is critical for staff and patients to understand that any significant change in clinical status or the onset or exacerbation of symptoms following in-office procedures, immunotherapy, or the ingestion of a known allergen should be considered anaphylaxis. Anaphylaxis should be regarded as a severe allergic reaction with rapid onset that can be fatal if not treated with epinephrine [8,9]. Regular inventory checks of the anaphylaxis cart (e.g., every three months) should be conducted, using a detailed checklist to ensure all supplies and medications are up to date [5].

To effectively manage outpatient emergencies, all practice settings where anaphylaxis risk exists should work with nursing staff to establish a customized written protocol. Various action plans for managing anaphylaxis in outpatient settings have been published [4]. These action plans should follow evidence-based guidelines and offer a clear, stepwise approach to treatment based on symptoms and patient response. The plan should be presented in an easily readable format, such as an algorithm, table, or graph, and should designate specific roles for staff members during an anaphylaxis emergency [19]. Once developed, this protocol should be posted in all patient care areas and alongside emergency supplies. Successful management of anaphylaxis requires that office staff activate the response team immediately and provide appropriate treatment promptly. Regular, organized mock anaphylaxis drills involving all staff, both clerical and medical, can help maintain preparedness [19]. Clinical proficiency in anaphylaxis management requires certification in basic cardiopulmonary resuscitation (CPR) and, ideally, advanced life support to ensure staff can manage refractory cases, including airway management, cardiac compressions, venous and intraosseous access, and medication delivery [19,22].

The initial evaluation and management of a patient experiencing anaphylaxis require several crucial steps that must be undertaken simultaneously [4,19,22]. Immediate intervention is necessary, as there is typically a limited time window (e.g., 5 minutes for iatrogenic intravenous allergens such as antibiotics and 30 minutes for food-induced anaphylaxis) before mild symptoms escalate to respiratory or cardiac arrest [23–27]. Although the ideal response involves removing the triggering allergen, this is often not feasible in clinical settings, as the allergen is usually administered or consumed prior to the onset of symptoms. However, during medication infusions or oral food or drug challenges, these procedures should be halted immediately upon the appearance of even mild anaphylactic symptoms [28,29].

The first medical personnel to recognize an anaphylactic reaction must promptly assess the patient's airway, breathing, circulation, and mental status [19]. In cases of moderate to severe anaphylaxis, or if signs of impending cardiopulmonary arrest are present, emergency medical services (EMS) must be contacted immediately, and all available medical staff should be mobilized [4,12]. Cardiopulmonary resuscitation (CPR) should commence without delay in the event of cardiac arrest, with an emphasis on continuous chest compressions [19,22]. Ventilation can begin once two medical personnel are present [22]. In instances of imminent or established cardiopulmonary arrest, intravenous access should be secured swiftly, and an intravenous bolus of epinephrine administered, despite the potential risk of ventricular arrhythmias following epinephrine use [19,22,30]. For adults, the intravenous dose is 1 mg (1:10,000 dilution), and it can be repeated every 3 to 5 minutes during ongoing CPR [22,31–33]. If intravenous access is unavailable, epinephrine can be administered endotracheally if an advanced airway is in place, with an adult dose of 2–2.5 mg (1:1000 dilution in 5–10 mL of sterile water) [19,22]. Management of anaphylaxis is generally based on indirect evidence and expert consensus [12]. Observational studies and analyses of near-fatal and fatal cases highlight the importance of prompt and decisive treatment with epinephrine to prevent progression to severe symptoms, even in mild cases [5,34]. Delayed administration of epinephrine is often cited as a major factor contributing to fatalities [5,34].

Immediate management steps include allergen removal (when possible), assessment of airway, breathing, circulation, and orientation, initiation of chest compressions at a rate of 100 per minute if cardiovascular arrest occurs, and intramuscular injection of 0.3–0.5 mg of epinephrine into the vastus lateralis [19,22]. Additional actions involve summoning appropriate assistance, positioning the patient (recumbent for adults and adolescents, left side for pregnant patients), administering 8–10 L/min of oxygen via face mask, and repeating intramuscular epinephrine injections every 5–15 minutes if there is no response to the initial dose (up to 3 doses) [19,22]. In the event of no immediate response or moderate to severe anaphylaxis, EMS should be activated, and intravenous fluids should be administered to manage hypotension or epinephrine non-response [19,22].

Additional treatments may include the administration of 2.5–5.0 mg of nebulized albuterol for lower airway obstruction, glucagon for patients on beta-blockers who do not respond to epinephrine (1–5 mg IV over 5 minutes), and epinephrine infusion for those unresponsive to initial intramuscular doses and saline infusions [19,22]. If intravenous access is not readily available, intraosseous access may be established for the administration of fluids and epinephrine [19,22]. For refractory cases, advanced airway management, including supraglottic airway placement, endotracheal intubation, or cricothyrotomy, may be required, particularly in cases of severe laryngeal edema or inadequate ventilation using a bag-valve mask [19,22]. If the patient remains unresponsive to initial treatment, dopamine may be administered, typically in a hospital setting with cardiac monitoring [19,22].

While treatments such as H1 antihistamines (e.g., diphenhydramine) and corticosteroids (e.g., methylprednisolone) can be considered, their efficacy in managing anaphylaxis remains unestablished, and they should not replace epinephrine as the primary intervention [19,22]. Patients should be transported to

the emergency department for further monitoring for at least 8 hours or observed in the office for an additional 30–60 minutes if not requiring emergency department care [19,22]. Post-recovery, patients should receive education on recognizing and managing anaphylaxis, including the prescription of autoinjectable epinephrine and a personalized action plan [19,22].

Summary of Adult Cardiac Life Support Recommendations

Basic Cardiopulmonary Resuscitation (CPR) follows the C-A-B sequence (Compressions-Airway-Breathing). It begins with checking the pulse, allowing a maximum of 10 seconds for assessment. Chest compressions should be performed forcefully and rapidly on the center of the chest, maintaining a rate of 100 compressions per minute. The depth of each chest compression should reach 5 cm, ensuring complete chest recoil after every compression. Interruptions during compressions must be minimized both in frequency and duration. Ventilations are performed only if two rescuers are present, avoiding excessive ventilation and confirming chest rise with each breath. The recommended duration of each ventilation is one second. If three rescuers are available, one focuses on compressions, while two others handle bag-valve-mask ventilation, rotating positions every two minutes. The compression-to-ventilation ratio is set at 30:2. When it comes to defibrillation, a single shock is delivered using the highest available energy (e.g., 200J for adults).
There are three distinct phases of resuscitation during cardiac arrest:

1. **Electrical Phase (0-4 minutes)**: This phase involves immediate defibrillation and chest compressions.
2. **Hemodynamic Phase (4-10 minutes post-arrest)**: Defibrillation and chest compressions are prioritized.
3. **Metabolic Phase (>10 minutes post-arrest)**: Very few patients survive after this stage.

Anaphylaxis Guidelines

Current anaphylaxis guidelines universally recommend administering epinephrine intramuscularly into the lateral thigh [4,5,14]. Research on epinephrine pharmacokinetics in individuals not experiencing anaphylaxis reveals that intramuscular injections in the vastus lateralis result in a quicker rise in blood epinephrine levels compared to subcutaneous or deltoid muscle injections [35,36]. Unfortunately, studies evaluating subcutaneous injections in the lateral thigh are lacking. For adults, the recommended dose of 1:1000 epinephrine is between 0.2 to 0.5 mL, while the pediatric dose is 0.01 mg/kg, up to a maximum of 0.3 mg [19]. For severe anaphylaxis, a higher dose (e.g., 0.5 mL) within the recommended range may be necessary [4,12,19]. If symptoms do not improve significantly, the dose can be repeated every 5 to 15 minutes as deemed appropriate by the physician. Up to 35% of patients may require an additional dose [37,38]. During treatment, it is essential to monitor the patient's blood pressure, heart rate, respiratory status, and oxygenation regularly [19]. Oxygen saturation monitoring should also be initiated, with continuous non-invasive monitoring, and an electrocardiogram (if available) should be obtained.

Although there is a consensus that patients experiencing anaphylaxis should be placed in a supine position [4,5,12,14,19,39,40], there is ongoing debate regarding whether to elevate the legs [4,41,42]. Some guidelines advocate for the Trendelenburg position (where the feet are elevated 15–30 degrees higher than the head) for managing shock. However, a 2010 consensus by the American Heart Association and the American Red Cross found insufficient evidence to recommend the Trendelenburg position for shock patients [30,43]. In cases where the patient experiences respiratory difficulty, sitting them up may be beneficial [30]. A retrospective study of 10 anaphylactic fatalities suggested that transitioning from a supine to an upright or standing position during anaphylaxis may be linked to fatal outcomes. However, the study did not support the use of the Trendelenburg position [44].

The administration of oxygen is the second most critical intervention for anaphylaxis, only behind epinephrine, and is recommended for all patients experiencing anaphylaxis, regardless of respiratory status [4,5,12,14,20,40]. Oxygen should be administered immediately to any patient with respiratory or cardiovascular compromise or those unresponsive to initial epinephrine treatment [19]. Oxygen at 100% concentration should be given at a flow rate of 6 to 10 L/min via a face mask. Oxygen saturation should be monitored and maintained between 94% and 96% [19]. In office settings, bag-valve-mask ventilation is the preferred method for supporting ventilation in cases of respiratory failure or arrest [4,12]. Two individuals are most effective in supporting the airway: one opens the airway using the head-tilt and chin-lift maneuver while sealing the mask over the nose and mouth, while the second person squeezes the bag, ensuring adequate chest rise [22]. Around 600 mL of tidal volume should be delivered over one second using a 1–2 L adult bag [22,30]. Supplementary oxygen should be supplied at a flow rate of 10 to 12 L/min [30]. In unconscious patients with no gag or cough reflex, an oropharyngeal airway may assist in delivering adequate ventilation [30]. The skill and experience of the attending physician should dictate the choice of the most appropriate airway device for each patient [22]. If ventilation can be sustained effectively using a bag-valve-mask, there is no evidence to support the use of advanced airway techniques to improve survival rates during out-of-hospital cardiac arrest [22,30,43]. When endotracheal intubation is performed by inexperienced providers, or when tube monitoring is insufficient, complications arise frequently [19]. Supraglottic airways (e.g., laryngeal mask airways, esophageal tracheal tubes, or laryngeal tubes) provide reasonable alternatives to endotracheal intubation and can be used without interrupting chest compressions [30]. For cases involving upper airway obstruction (e.g., severe laryngeal edema), endotracheal intubation is contraindicated [30]. Inhaled or intratracheal epinephrine may reduce oropharyngeal edema, easing airway management [19]. Cricothyrotomy should be reserved for life-threatening situations where upper airway obstruction (e.g., angioedema) prevents adequate ventilation [19,41].

Hypotension should be treated with rapid fluid replacement, administering 1 to 2 L of 0.9% normal saline (5–10 mL/kg within the first five minutes for adults, up to 30 mL/kg within the first hour for children) [22,30,45]. For adults, large-bore intravenous catheters (14-16 gauge) are recommended. For normotensive patients, maintaining normal saline at an appropriate rate (e.g., 125 mL/h for adults) to ensure venous access for medications or rapid fluid replacement may not always

be necessary [19]. Intravenous epinephrine is rarely required in outpatient settings and should only be administered in a monitored setting with a programmable infusion pump [4,12,19,40]. However, if multiple intramuscular injections of epinephrine and intravenous fluids fail, and there is a delay in EMS response, prolonged transport, or cardiopulmonary arrest, intravenous epinephrine may be necessary [19,30]. There is no standard dosage for intravenous epinephrine in anaphylaxis [4,8,12,20,40]. One prospective study demonstrated the effectiveness of administering a 1:100,000 epinephrine solution intravenously via an infusion pump at an initial rate of 2 to 10 mg/min, titrated based on clinical response or side effects [46]. If an epinephrine infusion is initiated in the outpatient setting, careful monitoring (e.g., blood pressure, pulse, and electrocardiography) must be in place, and preparations should be made to address potential ventricular arrhythmias [4,12,19,22,30]. Other vasopressors, such as dopamine and vasopressin, may serve as alternatives to epinephrine in cases of refractory hypotension, though no controlled studies have evaluated their efficacy for anaphylaxis [30,43].

Patients on beta-adrenergic blockers have been reported to experience unusually severe or resistant anaphylactic reactions, characterized by paradoxical bradycardia, significant hypotension, and intense bronchospasm [21,47]. These effects have also been observed with the use of ophthalmic beta-blockers [48]. The increased severity of anaphylaxis in these patients may be linked to a diminished response to epinephrine, commonly used for treatment [4,12]. When epinephrine is administered to patients on beta-blockers, there is a risk of unopposed alpha-adrenergic effects and reflex vagotonic responses, which could result in hypertension and potentially lead to cerebral hemorrhage [41]. Additionally, patients on beta-blockers may exhibit an increased risk of bronchospasm and reduced cardiac contractility, perpetuating hypotension and bradycardia. However, there are no epidemiological studies suggesting a higher frequency of anaphylaxis in individuals taking beta-blockers [49,50]. The use of selective beta1-antagonists does not mitigate the risk of anaphylaxis, as both beta1- and beta2-antagonists can block the beta-adrenergic receptor [12]. If epinephrine proves ineffective in managing anaphylaxis in patients on beta-blockers, glucagon may be necessary. Glucagon works by directly activating adenyl cyclase, bypassing the beta-adrenergic receptor, which can alleviate refractory bronchospasm and hypotension [12]. The recommended glucagon dose is 1 to 5 mg intravenously over five minutes, followed by an infusion of 5 to 15 mg/min, adjusted based on clinical response [4,12,30]. Protecting the airway is crucial, as glucagon may induce vomiting, posing a risk of aspiration in patients who are drowsy or unconscious. Placing the patient in a lateral recumbent position usually ensures adequate airway protection [4,12,19].

Antihistamines, both H1 and H2, are considered secondary treatments for anaphylaxis due to the lack of direct evidence supporting their efficacy in treating the condition [3-5,12,14,40]. The effectiveness of H1 antihistamines is largely inferred from their success in treating other allergic conditions, such as urticaria or allergic rhinitis, where they alleviate symptoms like itching, urticaria, flushing, sneezing, and rhinorrhea [12]. However, they do not address upper airway obstruction or hypotension. A common and often fatal error by healthcare professionals and patients is delaying the administration of epinephrine in

anticipation of symptom relief from antihistamines [8,20,22,39,51-54]. In severe anaphylaxis, sedating antihistamines like diphenhydramine are available for intravenous administration, with the adult dosage typically ranging from 25 to 50 mg, given intravenously over 10 to 15 minutes [19]. When given orally, non-sedating antihistamines, such as fexofenadine, are preferred to avoid drowsiness, cognitive impairment, and difficulty in symptom reporting [19]. If parenteral administration is needed, the dose of the H2 antihistamine ranitidine is 1 mg/kg for adults and can be given either intramuscularly or intravenously via slow infusion, as both methods have similar onset times [12,19].

Corticosteroids do not play a role in the acute treatment of anaphylaxis [22,30]. Although some evidence suggests corticosteroids may reduce the incidence of biphasic or prolonged reactions, this is not supported by robust data [8-10]. Their use and dosage are often based on protocols for acute asthma. When administered, the recommended intravenous or oral dose is 1 to 2 mg/kg per dose, up to 125 mg of methylprednisolone or an equivalent formulation [19]. Patients whose symptoms fully resolve after epinephrine administration do not require subsequent prescriptions for antihistamines or corticosteroids [4].

The length of direct observation and monitoring after an anaphylactic episode should be tailored to the individual, considering factors such as the severity and duration of the reaction, response to treatment, history of previous reactions, medical comorbidities, patient reliability, and access to healthcare [2,4,12,19,20,40]. Moderate to severe anaphylactic reactions generally warrant observation for at least four to eight hours [4,12,19]. For mild symptoms that resolve quickly with treatment in a medical setting, a shorter observation period may suffice. However, longer monitoring or even hospital admission should be considered if there are risk factors for severe anaphylaxis (e.g., a history of severe asthma), ingestion of allergens, a need for more than one dose of epinephrine, presence of pharyngeal edema, or prolonged or severe symptoms, such as persistent wheezing or hypotension [4,12,14,19,40,55,56].

Patient education is essential in managing anaphylaxis, particularly given the potential for rapid progression and life-threatening reactions. Patients who have experienced anaphylaxis or are at high risk should be prescribed autoinjectable epinephrine and educated on its proper use. It is critical that patients fill this prescription promptly, as up to 23% of cases may experience biphasic reactions within 10 hours after the initial resolution of symptoms [10]. Patients should be provided with two autoinjectors, as up to 30% of anaphylactic episodes require more than one dose of epinephrine [2,8,9,34]. In the United States, autoinjectors are available in two doses: 0.15 mg and 0.30 mg, with the latter being the preferred adult dose. While the initial anaphylaxis action plan can be developed in emergency or primary care settings, a long-term plan should be created in collaboration with an allergist, primary care physician, interdisciplinary clinical team, and, when necessary, the patient's school. Ongoing education on the triggers and early signs of anaphylaxis should be a structured and recurring process for both patients and healthcare staff [13].

Conclusion

Anaphylaxis represents a medical emergency requiring swift recognition and intervention to prevent fatal outcomes. This review highlights the critical role of epinephrine as the primary treatment for anaphylactic reactions. Intramuscular administration of epinephrine into the vastus lateralis ensures rapid absorption and efficacy, making it the first-line intervention in outpatient settings. While antihistamines, corticosteroids, and beta-agonists play supportive roles, they should never replace epinephrine as the mainstay of treatment. Effective management of anaphylaxis in outpatient settings demands comprehensive preparation, including staff education, regular drills, and maintaining a well-stocked emergency cart with essential supplies such as epinephrine autoinjectors, airway management tools, and intravenous fluids. Ensuring that staff are trained in cardiopulmonary resuscitation (CPR) and advanced life support can significantly improve outcomes, particularly in cases where the patient experiences severe respiratory or cardiovascular compromise. A key finding from this review is the importance of early intervention. Delayed epinephrine administration has been consistently linked to poor outcomes, underscoring the need for prompt and decisive action at the first signs of anaphylaxis. Additionally, regular mock drills and staff training sessions are essential for maintaining preparedness and ensuring that all team members are capable of executing the emergency protocol effectively. Beyond immediate treatment, patient education is crucial for preventing future incidents. Patients at risk of recurrent anaphylaxis should be equipped with autoinjectable epinephrine and provided with a personalized action plan. Follow-up care, including patient education on allergen avoidance and recognizing early symptoms, plays a pivotal role in long-term management. In conclusion, while the management of outpatient anaphylaxis is challenging, adherence to evidence-based protocols, rapid epinephrine administration, and a well-prepared staff can significantly improve patient outcomes. Ongoing education, preparedness, and clear emergency action plans are key to reducing the risk of fatal anaphylactic events in outpatient settings.

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مقالة مراجعة: طوارئ صدمة الحساسية المفرطة في العيادات الخارجية

الملخص:

الخلفية: تعتبر التأق (anaphylaxis) رد فعل تحسسي شديد ومهدد للحياة قد ينجم عن مواد مثل الطعام، أو الأدوية، أو لدغات الحشرات، أو العوامل البيئية. يظهر التأق مجموعة من الأعراض، بما في ذلك الأعراض التنفسية والقلبية الوعائية والجلدية والجهاز الهضمي، والتي يمكن أن تتطور بسرعة. يحدث التشخيص الخاطئ بشكل شائع نظرًا لتداخل الأعراض مع حالات مثل الصدمة الإنتانية أو الربو. تُعد الإدارة الفورية للأدرينالين عبر الحقن العضلي أمرًا حاسمًا للعلاج، إلى جانب إدارة المجرى الهوائي ومضادات الهيستامين والأستيرويدات القشرية.

الهدف: تهدف هذه المراجعة إلى استكشاف إدارة صدمة الحساسية المفرطة في العيادات الخارجية، مع التركيز على الأعراض واستراتيجيات العلاج والاستعدادات الطارئة. تركز المراجعة على أهمية التدخل السريع وتدريب الموظفين لإدارة ناجحة.

الطرق: تتضمن الطرق تحليل الأدبيات الحالية المتعلقة بالتأق، وتقديمه السريري، وبروتوكولات العلاج الطارئ.

النتائج: أظهرت النتائج أن الإدارة المبكرة للأدرينالين أمر بالغ الأهمية لتحسين النتائج، مع مساهمة التأخير في العلاج في زيادة مخاطر الوفيات. يُعد الاستعداد المنتظم، مثل الحفاظ على عربة تأق وتنفيذ تدريبات للموظفين، أمرًا حيويًا للرعاية الفعالة في العيادات الخارجية.

الخلاصة: تؤكد الخلاصة على ضرورة تجهيز العيادات الخارجية بالبروتوكولات المناسبة والإمدادات الطارئة للتعامل مع حالات الطوارئ صدمة الحساسية المفرطة، وكذلك الحاجة إلى تثقيف المرضى حول التعرف على نوبات التأق المستقبلية وإدارتها.

الكلمات المفتاحية: الحساسية المفرطة، طوارئ العيادات الخارجية، الأدرينالين، إدارة المجرى الهوائي، الغلوبولين المناعيE ، رد الفعل التحسسي، الصدمة، الاستعداد للطوارئ.