# Anaphylaxis - Management of Suspected Anaphylaxis in Adults. Clinical Guideline

Reference no.: CG-T/2023/011

#### Introduction

The incidence of anaphylaxis in the UK is on the rise, with a reported increase in hospital admissions for anaphylaxis from 1 to 7 cases per 100,000 of the population per year between 1992 and 2012. An estimated 20 deaths from anaphylaxis are reported each year in the UK (1).

NICE and the Resuscitation Council UK have published guidelines on the management of suspected anaphylaxis in adults, with which the current guidelines are compliant (1,2). The World Allergy Organisation (WAO) have also published comprehensive guidelines on anaphylaxis (3).

The Adult Allergy Clinic runs on Wednesday mornings at The Royal Derby Hospital. Referrals for suspected anaphylaxis can be made using the form found in appendix 1. This should be emailed to <u>dhft.RespiratorySecretaries@nhs.net</u>. Referrals for anaphylaxis secondary to drugs are NOT accepted.

#### Purpose and Scope of Guideline

The purpose of this clinical guideline is to ensure that all adult patients suspected of suffering an anaphylactic reaction are given prompt, effective treatment by an appropriately trained healthcare professional, in line with the Resuscitation Council UK and NICE guidelines on anaphylaxis. The clinical guideline applies to all clinical and non-clinical areas within the Trust. Any healthcare practitioner can initiate the early treatment of a person suffering an anaphylactic reaction, whatever the cause (this includes approved non-professional groups as well as practitioners recognised under the Medicines Act).

In addition to the emergency management of anaphylaxis (section 4.5) section 4 of the guideline also covers the other aspects of suspected anaphylaxis management, as outlined below.

- 4.1 What is anaphylaxis?
- 4.2 Common causes of anaphylactic and anaphylactoid reactions.
- 4.3 Co-factors and risk factors in anaphylaxis.
- 4.4 Differential diagnosis of anaphylaxis.
- 4.5 Treatment of anaphylaxis.
- 4.6 Investigations.
- 4.7 Documentation.
- 4.8 Patient education and adrenaline auto-injectors.
- 4.9 Safe discharge.

- 4.10 Referral to the Adult Allergy Clinic.
- 4.11 Peri-operative anaphylaxis.

#### Definitions

ACE: angiotensin converting enzyme; IM: intramuscular; IV: intravenous; NICE: National Institute for Health and Care Excellence; NSAID: non-steroidal anti-inflammatory drug

#### Main body of Guidelines

#### 4.1 What is anaphylaxis?

Anaphylaxis is a potentially life threatening reaction which usually occurs in response to exposure to an allergen against which the patient has previously developed IgE antibodies (i.e. become sensitised to). In general, symptoms develop within minutes to a few hours of exposure to the culprit allergen and can be rapidly progressive. The WAO has defined anaphylaxis as being highly likely when any of the following two criteria are met (3):

1. Acute onset of an illness (minutes to several hours) with simultaneous involvement of the skin, mucosal tissue, or both (e.g., generalised hives, pruritus or flushing, swollen lips, tongue, uvula).

AND AT LEAST ONE OF THE FOLLOWING:

- a. Respiratory compromise (e.g., dyspnoea, lower airway wheeze, stridor, reduced peak expiratory flow, hypoxaemia).
- b. Reduced blood pressure or symptoms of end-organ dysfunction (e.g., 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<sup>a</sup> or bronchospasm<sup>b</sup> or laryngeal involvement<sup>c</sup> after exposure to a known or highly probable allergen for that patient (minutes to several hours), even in the absence of typical skin involvement.

<sup>a</sup> decrease in bp more than 30% from patient's baseline or less than 90 mmHg systolic. <sup>b</sup> excluding lower respiratory symptoms triggered by common inhalant allergens or food allergens perceived to cause "inhalational" reactions in the absence of ingestion. <sup>c</sup> Stridor, vocal changes, or odynophagia

Anaphylactoid reactions are discussed in 4.2.

Anaphylaxis arises as a result of the rapid release of chemical mediators from mast cells. In most cases, sensitisation to the involved allergen will have occurred at some point in the past. Sensitisation involves the formation of IgE antibodies (specific to an allergen), which then bind to mast cells around the body. Upon subsequent re-exposure, the allergen binds to those IgE antibodies which recognise it, which leads to rapid mast cell degranulation and release of chemical mediators, causing anaphylaxis. The hallmark of an IgE mediated reaction is that symptoms occur within minutes to a few hours of exposure to the allergen and are usually rapidly evolving. Some rare exceptions to this are discussed in 4.2.

Anaphylaxis represents the most severe form of an IgE mediated reaction. Some patients may present with less severe symptoms. These patients should be referred to the allergy clinic for an assessment, and prescription of an adrenaline auto-injector considered. Severity of one reaction does not predict the severity of subsequent reactions.

In approximately 5% patients a biphasic reaction may occur, in which a recurrence of symptoms typically occurs some four to six hours later (can be up to 72 hours) (2,3). This is due to the recruitment of peripheral blood eosinophils to the affected organs.

#### 4.2 Common causes of anaphylaxis and anaphylactoid reactions

In children and young adults, foods are the most common cause of IgE mediated allergic reactions. In one study of young adults presenting with new onset food allergy, 49% experienced anaphylaxis and the commonest culprit foods were tree nuts, peanuts, soya, shellfish and fish, although many other foods are implicated (4). In older adults Hymenoptera insect venom IgE mediated allergy, most commonly to bees and wasps, and drug allergy, are the most common precipitants of anaphylaxis. Immediate reactions to drugs may be IgE mediated, for example to antibiotics, or non-IgE mediated, for example to NSAIDs, opiates and synthetic colloid fluid replacement products (5). The latter are termed anaphylactoid reactions as they do not require prior exposure to the drug and sensitisation through the production of IgE antibodies. NSAIDs are thought to cause anaphylactoid reactions by leading to excess leukotriene production in susceptible people but in some cases, reactions can be IgE mediated. Opiates can cause the direct release of histamine from mast cells. To all intents and purposes however the clinical presentation is the same and patients should be managed in the same way. IgE mediated reactions to natural rubber latex are now less common but are still an important cause of anaphylaxis, particularly in the healthcare setting (3).

Rarely, anaphylaxis may be caused by exercise or by the combination of exercise and ingestion of a food protein against which the patient has specific IgE antibodies. In food-dependent exercise induced anaphylaxis there may be a delay of some hours following ingestion before anaphylaxis develops and the reasons for this are incompletely understood (6). Similarly, some patients with IgE antibodies to certain plant food proteins (lipid transfer proteins) may only react if a co-factor such as exercise, stress, non-steroidal anti-inflammatory drug (NSAID) or alcohol ingestion or inter-current infection, is present in addition to food ingestion. These reactions may also be delayed by several hours after allergen exposure (7).

Following investigation in the allergy clinic the patient may be diagnosed with idiopathic/spontaneous anaphylaxis where no culprit allergen can be found.

#### 4.3 Co-factors and risk factors in anaphylaxis

Certain co-factors may reduce the threshold for developing anaphylaxis following allergen exposure or increase the severity of the reaction. Co-factors include infection, alcohol consumption, NSAID ingestion, exercise, emotional stress and pre-menstrual hormone changes in women (3).

The presence of asthma, particularly if uncontrolled, increases the risk of fatal anaphylaxis. Age increases the likelihood of fatality, and this is likely related to the presence of cardiovascular disease and/or drugs such as beta-blockers and angiotensin converting enzyme (ACE) inhibitors. Young adults may display greater risk-taking behaviour, for example failing to carry an adrenaline auto-injector (3).

## 4.4 Differential diagnosis of anaphylaxis

Table 2 outlines the important differential diagnoses of anaphylaxis. This list is not exhaustive.

Diagnosis	Distinguishing features	Notes
ACE inhibitor induced angioedema	Angioedema of lips, tongue and upper airway are common. Angioedema may affect any other mucosa, such as gut or genitalia. No urticaria or other features of anaphylaxis.	Common. No tests needed. Stop ACE inhibitor. May take several months to settle.
C1 esterase inhibitor deficiency	Angioedema. Urticaria is not a feature- if present this is not the diagnosis.	Rare. Inherited or acquired. Uncommon. Request C3 and C4 levels. If C4 reduced lab will test for C1 esterase levels and function.
Chronic spontaneous urticaria +/- angioedema	Recurrent urticaria +/- angioedema occurring for longer than 6 weeks. Rarely causes hypotension. Most common presentation is recurrent itchy, non-painful urticarial wheals each lasting less than 24 hours, with or without angioedema.	Classically the patient has searched for an allergic cause without success. Condition is idiopathic or in some cases autoimmune, i.e., there is no allergic basis. Waking with symptoms effectively excludes food allergens as a cause. Instruct patient to take a daily non-sedating antihistamine and in severe cases a short course of oral prednisolone may be required. Patient should see GP for further assessment and referral to dermatology or allergy clinic considered to rule out other causes and aid long term management. Avoid ACE inhibitors if patient has experienced angioedema.
Vasovagal reaction (fainting)	Classically causes low blood pressure and loss of consciousness with an initial slow rather than fast pulse rate. Urine incontinence may be a feature. Responds quickly to lying the patient flat.	Absence of other features such as skin rash, angioedema, respiratory features etc.
Panic attack	Presents with anxiety and sense of impending doom, shortness of breath, dizziness	No wheeze, stridor, urticaria or angioedema. No hypotension.

#### Table 2 Differential Diagnoses of Anaphylaxis.

and weakness due to

	hyperventilation. Flushing or blotchy skin may be present.	
Septic shock	Presents with hypotension and tachycardia. Fever and peripheral vasodilation often present. In some cases, a petechial and/or purpuric rash may be present. History and examination findings will often guide to the source of sepsis.	Petechiae are small bleeds beneath the surface of the skin, measuring less than 3mm. Purpura are larger bleeds beneath the skin, measuring 3 to 10mm in diameter. Both are non- blanching. Can occur in the context of meningococcal septicaemia and sepsis associated with disseminated intravascular coagulation.
Phaeochromocytoma	Presents with attacks of headache, palpitations, sweating, elevated blood pressure. May have abdominal pain, tremor, anxiety and sense of doom. Urticaria, angioedema, wheeze, hypotension not a feature (postural hypotension may be).	Rare catecholamine secreting tumour.
Carcinoid syndrome	Episodic flushing, often of the face, diarrhoea, abdominal pain, palpitations, wheezing. A severe crisis may cause hypotension.	Rare. Caused by hormones released from metastatic carcinoid tumours.
Inducible laryngeal obstruction (vocal cord dysfunction)	Recurrent attacks of rapid onset difficulty in breathing, sensed in upper chest or throat and worse on inspiration. Paradoxical closure of vocal cords on inspiration. Triggers include acid reflux, post-nasal drip, emotional stress.	Often coexists with difficult asthma. Same pattern of symptoms each episode, in contrast with anaphylaxis.

## 4.5 Treatment

Figure 1 shows the Resuscitation Council UK treatment algorithm for anaphylaxis (2021 version), which should be used in all cases of suspected anaphylaxis. **The key points from this are management of the patient using an ABCDE approach with prompt administration of intramuscular adrenaline, whilst calling for senior help.** Adrenaline will treat the oedema and vasodilation leading to airway, breathing or circulatory dysfunction, any of which can be fatal. Intramuscular (IM) adrenaline should be administered as soon as anaphylaxis is recognised and **repeated after 5 minutes** if there is inadequate response.

In a case series of 92 patients who died from anaphylaxis only 23% received adrenaline prior to cardiorespiratory arrest, highlighting the probable importance of this treatment in

reducing the risk of death (8). Adrenaline induces more forceful and faster heart contractions, via beta-1 receptors, and vasoconstriction via alpha-1 receptors, preventing or reversing shock. Vasoconstriction also reduces oedema, including in the upper airway. In addition, adrenaline causes bronchodilation in the lower airway via its effects on beta-2 receptors and increases the rate and force of heart contractions via beta-1 receptors (3).

Common side effects of adrenaline include pallor (due to vasoconstriction), anxiety, palpitations, tremor and headache. More serious adverse effects, such as ventricular arrhythmias or pulmonary oedema, particularly in patients with underlying heart disease, have been observed in patients who received incorrect dosing and via the intravenous route (3). It is vital, however, that patients with anaphylaxis are not denied prompt administration of IM adrenaline. To this end all clinicians should be familiar with the dosing and administration of IM adrenaline as outlined below and in figure 1.

Following administration of the first dose, measure vital signs (respiratory rate, oxygen saturations, heart rate, BP, level of consciousness) and auscultate for wheeze to monitor the effect of treatment. *Repeat* the IM adrenaline dose after 5 minutes if there is no improvement in the patient's condition.

#### 4.5.1 Administration of Adrenaline

Intramuscular adrenaline Is the first line treatment for anaphylaxis. Adrenaline should be administered by a healthcare practitioner trained in the administration of IM injections and in line with Trust policy. The best site for IM injection is the anterolateral aspect of the middle third of the thigh. Adrenaline 1:1000 solution for intramuscular injection should be available in 1mg in 1mL ampoule (s) in the anaphylaxis blue box in clinical areas.

# Dose (Adult): Adrenaline 500 micrograms (0.5 ml) by IM injection. A blue 23G/25mm needle is suitable for most patients. Consider use of a green 21/38mm needle in patients with obesity.

The administration of intravenous adrenaline may be used cautiously by medical staff trained in using it in <u>non-cardiac arrest patients</u> and in specific clinical areas with higher levels of patient monitoring e.g., critical care.

#### 4.5.2 Removal of the trigger

When the suspected cause of anaphylaxis is an intravenous drug or intravenous infusion the infusion or injection should be stopped immediately and disconnected from the patient. In bee venom induced anaphylaxis it may be possible to remove the stinger. Do not delay definitive treatment if removing the trigger is not feasible.

#### 4.5.3 Lie the patient flat and raise legs

Cardiac arrest can occur suddenly if the patient stands, walks or sits up suddenly during the acute phase of a reaction. Patients must NOT walk or stand during this phase. Lying the patient flat and raising the legs is helpful for low blood pressure. Patients with airway or breathing problems may prefer to be in a semi-recumbent position. Patients who are breathing normally but unconscious should be placed on their side (recovery position). Pregnant patients with a palpable uterus (usually from 20 weeks) should lie on their left-hand side to prevent aortocaval compression. Head-down positioning of the bed should be performed rather than raising the legs.

#### 4.5.4 Oxygen

Give high flow oxygen at 15L/minute via mask and reservoir bag. Oxygenation is likely to be compromised in patients with upper airway obstruction or bronchospasm and difficult to rule out rapidly with a saturation monitor in patients with poor peripheral perfusion due to hypotension. Perform arterial blood gas testing once the patient is stabilised, aiming for saturations of 94-98% in most. Aim for 88-92% in patients with chronic hypercapnic respiratory failure or a past medical history that puts them at risk of hypercapnic respiratory failure if over-oxygenated (e.g., severe COPD).

#### 4.5.5 Intravenous (IV) fluids

In the presence of hypotension/ shock or poor response to an initial dose of adrenaline, establish IV access and give a fluid challenge of 500-1000ml of 0.9% saline or Hartmann's solution. If hypotension persists, further doses of adrenaline are likely to be required, together with further crystalloid. Hartmann's solution is preferred over 0.9% saline, as this is less likely to cause hyperchloremia. Senior Emergency or Adult Resuscitation Team staff should be present.

#### 4.5.6 Antihistamines

Antihistamines are *not* recommended as part of the initial emergency management for anaphylaxis. They have no role in treating respiratory or cardiovascular symptoms of anaphylaxis. Antihistamines can be used to treat cutaneous symptoms that may occur as part of allergic reactions, including anaphylaxis, but must not be given in preference to adrenaline to treat anaphylaxis. Consider using a non-sedating oral antihistamine (e.g., cetirizine) only once the patient has been stabilised.

#### 4.5.7 Corticosteroids

The routine use of corticosteroids to treat anaphylaxis is no longer advised. Consider giving steroids only after initial resuscitation for refractory reactions or ongoing asthma symptoms. Steroids must not be given preferentially to adrenaline.

Corticosteroids will not affect the severity of the initial reaction but may reduce the recruitment of eosinophils, thereby reducing the severity of any late phase reaction. Corticosteroids may be indicated where an acute asthma exacerbation may have contributed to the severity of anaphylaxis. Steroids should be given by the oral route where possible.

#### 4.5.8 Bronchodilators

If the patient is wheezy, in addition to first line treatment, consider inhaled bronchodilator therapy. Give oxygen driven nebulised salbutamol (2.5-5mg) and ipratropium bromide (500 micrograms). Bronchodilators should *not* be used as an alternative to further parenteral treatment with adrenaline in the presence of persisting respiratory problems. Magnesium sulphate can cause significant vasodilatation, worsening hypotension, so should not be given routinely (2).

#### 4.5.9 Refractory Anaphylaxis

Refractory anaphylaxis is defined as anaphylaxis requiring ongoing treatment (due to persisting respiratory or cardiovascular symptoms) despite two appropriate doses of IM adrenaline (2). When refractory anaphylaxis occurs critical care support should be sought early.

Maintenance adrenaline therapy is critical, using a low dose IV adrenaline infusion (see Figure 2 Resuscitation Council UK Treatment Algorithm for Refractory Anaphylaxis). IV adrenaline should be given only by experienced specialists in an appropriate setting. If an IV adrenaline infusion cannot be administered immediately continue to give IM adrenaline after every 5 minutes while life threatening cardiovascular and respiratory features persist.

Adrenaline therapy should be supported with fluid resuscitation. Give further fluids as necessary. A large volume (up to 3-5 litres in adults) may be required for severe anaphylactic shock.



Suitable for printing to guide individual patient management but not for storage

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Figure 2: Resuscitation Council UK Treatment Algorithm for Refractory Anaphylaxis

Resuscitation Council UK

GUIDELINES



Expiry date:Dec 2026

#### 4.6 Investigations

Tryptase is one of the mediators released from mast cells and is measurable in the laboratory. It rises rapidly after the onset of anaphylaxis, peaking at 1-2 hours and then falls to baseline. A raised tryptase supports the diagnosis of anaphylaxis. A value within the normal range does not exclude anaphylaxis.

A serum tryptase should be taken as soon as possible after presentation but should not delay emergency treatment. A second sample should be taken within 1-2 hours but no later than 4 hours after symptom onset (yellow top bottle).

#### 4.7 Documentation

Accurate documentation of events is essential (1). Ambulance Service documentation and Emergency Department observation charts and notes should also be uploaded to the local electronic patient record for future access.

A list of the key points for documentation is outlined below.

- Symptoms and signs.
- Recorded observations (blood pressure, pulse, oxygen saturations etc) at presentation and following treatment.
- Time of onset of reaction.
- Circumstances preceding onset of symptoms (may help identify trigger).
- Treatment given by paramedics and/or the Emergency Department.

#### 4.8 Patient Education and Adrenaline Auto-injectors

Offer patients and their family information about the following points:

- Information about anaphylaxis, including the signs and symptoms of an anaphylactic reaction.
- Information about the risk of a biphasic reaction.
- What to do if an anaphylactic reaction occurs (use the adrenaline auto-injector and call emergency services. Take an antihistamine if possible).
- A <u>demonstration</u> of the correct use of the adrenaline injector and when to use it (trainer auto-injectors are available for demonstration and training). <u>Prescribe two</u> <u>adrenaline auto-injectors with advice to carry them at all times, from the point</u> <u>of hospital discharge.</u>
- Advice about how to avoid the suspected trigger (if known).
- A referral will be made to the Adult Allergy clinic at The Royal Derby Hospital or elsewhere if appropriate.
- Patient support group: Anaphylaxis UK (<u>http://www.anaphylaxis.org.uk/</u>)

Two adrenaline auto-injectors should be prescribed in most cases of suspected anaphylaxis. An auto-injector is generally not necessary in drug-induced anaphylaxis as the culprit drug should be easier to avoid than food or insect triggers. Prescribe an auto-injector if you are unsure whether a drug was the sole trigger for the allergic reaction. A decision about the need for a life-long auto-injector can be made in the Allergy clinic.

EpiPen® (MEDA) and Jext® (ALK Abello) are the most commonly prescribed adrenaline autoinjectors. The dose in adults is 300 micrograms. User guides and demonstration videos are available on the company websites (see section 5 for links). Advise the patient to carry non-sedating antihistamines in addition to both adrenaline auto-injectors.

#### 4.9 Safe Discharge

The optimum time period for observation in hospital after an episode of anaphylaxis is unknown. NICE suggest 6 to 12 hours post onset of symptoms in general (1). The Resuscitation Council UK suggest the observation periods shown in figure 3 (2).

# Prior to discharge all patients should be assessed for the presence of dizziness on standing and lying/standing or sitting/standing blood pressures measured if present.

Consider fast-track discharge (after 2 hours observation from resolution of anaphylaxis) if:	Minimum 6 hours observation after resolution of symptoms recommended if:	Observation for at least 12 hours following resolution of symptoms if any one of the following:	
<ul> <li>Good response (within 5–10 minutes) to a single dose of adrenaline given within 30 minutes of onset of reaction         <ul> <li>Complete resolution of symptoms</li> <li>Complete resolution of symptoms</li> <li>The patient already has unused adrenaline auto-injectors and has been trained how to use them</li> <li>There is adequate supervision following discharge</li> </ul> </li> </ul>	<ul> <li>2 doses of IM adrenaline needed to treat reaction*</li> <li>or</li> <li>Previous biphasic reaction</li> </ul>	<ul> <li>Severe reaction requiring &gt;2 doses of adrenaline.</li> <li>Patient has severe asthma or reaction involved severe respiratory compromise.</li> <li>Possibility of continuing absorption of allergen, e.g. slow-release medicines.</li> <li>Patient presents late at night, or may not be able to respond to any deterioration.</li> <li>Patients in areas where access to emergency care is difficult.</li> </ul>	
In all cases, discharge must comply with NICE Clinical Guidance CG134.96			

Figure 3: Resuscitation Council UK risk stratification approach to length of in-hospital observation.

\*It may be reasonable for some patients to be discharged after 2 hours despite needing two doses of IM adrenaline, e.g. following a supervised allergy challenge in a specialist setting.

#### 4.10 Referral to the Adult Allergy Clinic

Please refer cases of non drug-related suspected anaphylaxis to the Adult Allergy Clinic at the Royal Derby Hospital. A referral form can be found in appendix 1.

Allergic reactions with IgE mediated features (e.g., urticaria; angioedema; lip or tongue swelling and itch), but not fulfilling the criteria for anaphylaxis should still be referred to the clinic for further management. Appropriate investigation in the clinic, allergen avoidance advice and adrenaline auto-injector training may help to prevent the patient experiencing anaphylaxis in the future.

**Anaphylaxis due to suspected drug allergy** should not be referred to the general allergy clinic at Derby. Regional centres with specific Drug Allergy Clinics include Nottingham University Hospitals (Queens Medical Campus), Glenfield Hospital and Heartlands Hospital, Birmingham. Ask the patient's GP to refer to the nearest centre and provide patient with documentation of the events as outlined in section 4.7. Please refer to NICE Drug allergy: Diagnosis and Management guideline for further guidance on drug allergy (9).

#### 4.11 Peri-operative Anaphylaxis

Peri-operative anaphylaxis is an unexpected critical event presenting suddenly and without warning (often much faster than other causes of anaphylaxis). It may occur in patients with no chronic health problems. In extreme cases, there is rapid progression to cardiopulmonary arrest, which may be fatal despite prolonged attempts to resuscitate the patient. The most common triggers include antibiotics (penicillin, teicoplanin), muscle relaxants, chlorhexidine and patent blue dye. The treatment of peri-operative anaphylaxis should follow the standard algorithms. It should be noted that the timescale of deterioration is often more rapid than other causes of anaphylaxis, and over 75% of patients require ITU admission.

The anaphylaxis lead for the anaesthetic department is Dr P Tandon. Please email details of all cases of peri-operative anaphylaxis to puneesh.tandon@nhs.net. Help with the referral pathway to the nearest Drug Allergy clinic can be offered, including documentation requirements. Please visit The Royal College of Anaesthetists website, National Audit Project 6 for further information (10).

#### **References/Links**

- 1. Anaphylaxis: assessment and referral after emergency treatment. Published: 14 December 2011.Updated 2021. <u>nice.org.uk/guidance/cg134</u>
- Resuscitation Council UK. Emergency treatment of anaphylactic reactions. Guidelines for healthcare providers. <u>https://www.resus.org.uk/anaphylaxis/emergency-treatment-of-anaphylactic-reactions/</u>
- 3. World Allergy Organization Anaphylaxis Guidance 2020. Published:October 30, 2020 DOI:https://doi.org/10.1016/j.waojou.2020.100472
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- 8. Xu YS, Kastner M, Harada L, Xu A, Salter J, Waserman S. Anaphylaxis-related deaths in Ontario: a retrospective review of cases from 1986 to 2011. Allergy Asthma Clin Immunol. 2014;10:38.
- 9. Drug allergy: diagnosis and management (https://www.nice.org.uk/guidance/cg183)
- 10. <u>NAP6: Perioperative Anaphylaxis The National Institute of Academic Anaesthesia</u> (nationalauditprojects.org.uk)

Links to adrenaline auto-injector user guides:

http://www.epipen.co.uk/patients/epipenr-user-guide/

https://kids.jext.co.uk/about-jext/how-to-use/

#### **Documentation Controls**

Development of Guideline:	Jennie Gane, Respiratory Consultant
Consultation with:	Ed, Respiratory
Approved By:	Med Division – December 2023
Review Date:	December 2026
Key Contact:	Jennie Gane

#### Appendices

Appendix 1- Suspected Anaphylaxis Referral Form

Appendix 2- MHRA Information leaflet: "The correct use of your adrenaline auto-injector

#### Suspected Anaphylaxis Referral Form (Adults)\*

*Anaphylaxis caused exclusively by a drug should be		
referred to a regional drug allergy clinic		
(QMC/Glenfield/Birmingham Heartlands). DO NOT refer		
to general allergy clinic. Please see guidelines.		

Patient name:	
D/NHS number:	••
DOB:	
Address:	

Date of reaction: .....

Referral source: .....

Symptoms	and	clinical	signs:
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		 •••••	 
Emergency trea	itment delivered:		
	/ <b>.</b>		

#### Possible trigger (state if unknown):

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Management of patients meeting criteria for Suspected Anaphylaxis	Tick when complete
Tryptase sample on arrival*	
Second tryptase sample 1-2 hours later*	
Adrenaline auto-injector x2 dispensed	
Advice and guidance given (see below)	
*not later than 4 hours post reaction	

#### Advice on Discharge for Patients with Suspected Anaphylaxis

- 1. Explain the symptoms of anaphylaxis
- 2. Provide the patient with <u>two</u> adrenaline auto-injectors (<u>unless certain anaphylaxis was exclusively</u> <u>due to a drug reaction</u>)
- 3. Show them how to use it and signpost to company on-line training videos
- 4. When to use it (wheeze, SOB, throat closure, feeling faint, abdominal pain or vomiting in presence of other allergic symptoms)
- 5. What to do if anaphylaxis occurs (print appendix 2: MHRA "Correct use of your AAI" leaflet)
- 6. Risk of a biphasic reaction
- 7. Trigger avoidance if relevant
- 8. Offer referral to RDH Allergy clinic and advise them of the following patient support group: Anaphylaxis UK (http://www.anaphylaxis.org.uk/)

Please email a completed referral to dhft.RespiratorySecretaries@nhs.net or send in the post to the Respiratory Secretaries, Medicine Office Suite A, Off Ward 408, Level 4, RDH. Burton site referrals: please enclose copies of the ED notes and ambulance paperwork. Referrals without adequate clinical information will be returned.