Anaphylaxis

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Anaphylaxis is a severe, life-threatening, generalised or systemic **hypersensitivity** reaction, with significant disturbance of one or more of airway, breathing or circulation. It is not clear why one person with specific **immunoglobulin E** (**IgE**) to an **allergen** will have an anaphylactic reaction on exposure, another only a local reaction, and in a third individual no reaction at all. Some risk factors have been defined, such as low levels of platelet activating factor acetylhydrolase and low levels of serum angiotensin converting enzyme, both of which independently increase the risk of an allergic individual developing anaphylaxis on allergen exposure. Local and systemic allergic reactions occur via similar mechanisms that differ in location and magnitude. It should be noted that fatal allergic reactions can occur without anaphylaxis being present. For example, angioedema affecting the upper airway may be a lethal local reaction and other reactions may kill by inhalation of vomit. Some medicines such as non steroidal anti-inflammatory drugs (NSAIDS) can worsen allergic reactions including anaphylaxis.

Anaphylaxis results from the actions of a wide range of mediators released by **mast cell** and **basophil** degranulation (**Table 1**). Many of these mediators are preformed and stored in the granules, whereas others are produced *de novo* on activation of mast cells and basophils. Degranulation can be mediated by cross-linking of IgE bound to **membrane high-affinity IgE receptor** (**FcεRI**), or by non-IgE-mediated mechanisms. The distinction between these mechanisms can be important diagnostically, but their clinical presentation and the medical management of the acute emergency they cause are indistinct.

Table 1. Examples of mediators released during anaphylaxis (adapted from Immunobiology, Janeway et al)

The clinical presentation of anaphylaxis is variable and many different organ systems may be affected. The skin may itch (pruritus) with or without weals (urticaria) and/or swelling (angioedema). There may be nausea, abdominal pain, vomiting and/or diarrhoea. Swelling may involve the lip, tongue, throat and/or upper airway impairing swallowing (dysphagia), speech (dysphonia) or breathing (with stridor and/or asphyxiation). The lungs can be affected with cough, wheeze and bronchospasm with a corresponding fall in the peak expiratory flow rate. Cardiovascular events include chest pain, hypotension and fainting (syncope).

The emergency treatment of anaphylaxis involves the prompt administration of **adrenaline**. Other treatments such as anti-histamines, intravenous fluids and steroids are also commonly used, but should not lead to a delay in the administration of adrenaline. Adrenaline autoinjectors are commonly prescribed to patients at high risk of anaphylaxis, so that they are able to self-administer adrenaline in an emergency (**Figure 1**). After surviving an episode of anaphylaxis, it is important that the patient is referred to an Immunology or allergy clinic to identify the cause, and thereby reduce the risk of future reactions and prepare the patient to manage future episodes.

Figure 1. An example of an adrenaline autoinjector