

## The Aetiology and Pathophysiology of Anaphylaxis!

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### **Short Commentary**

There appears to be a complex relationship between eosinophils, mast cells (tissue basophils) and dorsal horn receptor pruritus cells in the aetiology of anaphylactic or anaphylactoid reactions.

The most important cell is the mast cell as this cell releases histamine and tryptase which causes bronchospasm and ultimately results in anaphylaxis.

But most people with allergic diatheses have a high eosinophil count. This usually matches the IgE level.

Eosinophils release eosinophil chemotactic factor and eosinophil cationic protein.

The eosinophil count in helminthic infestations is usually higher than in allergic diatheses.

The dorsal horn cell in the spinal cord have pruritic receptors that cause itching and react to histamine and tryptase release from the mast cells.

So there is a complex relationship between eosinophils, mast cells and dorsal horn pruritic receptor cells.

Chemotactic factors such as hymenoptera stings, peanut allergy and other allergens initiate a cascade of events that result ultimately in the demise of the patient unless adrenalin in the form of epinephrine is given. Antihistamines and steroids may also be of some help.

Severe asthma is a type of anaphylactic reaction and here the bronchospasm must be broken.

The same complex relationship between the mast cell, the eosinophil and the dorsal horn pruritic receptor cell applies. There is a complex feedback mechanism that down regulates and controls the intricate events that lead to anaphylaxis and ultimately death!

Post mortem histology on patients who have died of anaphylaxis show marked bronchospasm, pulmonary oedema and severe congestive cardiac failure with high eosinophil counts in the congested lungs. Often this is found after ingestion of shellfish, gluten, pork etc..

The adrenalin can be given intramuscularly if necessary and theophyllin derivatives may be of some value. Leukotrienes have been implicated as having complement and properdin!