

Asthma: Pathogenesis

Genetic factors

(i.e. HLA gene mutations, defects in bronchial airway epithelium)

Environmental factors

(i.e. excess hygiene, fewer siblings, antibiotics within the first two years)

Atopy:

predisposition to allergic hyper-sensitivity in airways

First exposure to triggers* sensitizes helper T cells

Stimulation of B-cells to produce IgE, which binds to mast cell surfaces

Activated Helper-T cells & IgE-sensitized mast cells now line the airways

Second exposure to triggers

Early response (0-2 hrs)

Allergens cross-link IgEs on mast cells

Mast cells release histamines, leukotrienes, and other inflammatory mediators

Vascular permeability → edema of airway mucosa

Goblet cell hyperplasia → ↑ mucus secretion

Bronchial smooth muscle contraction

Airway obstruction

Delayed response (3-4 hrs)

Activated mast cells & helper T cells release cytokines

Induce maturation of granular WBCs like eosinophils

Eosinophils migrate into:

Airways

→ Bronchiole constriction

Eyes

→ Conjunctivitis

Nose

→ Rhinitis

Asthma

Note: Delayed response presents within 3-4 hrs, peaks within 6-8 hrs, and resolves within 24 hrs

Asthma:
Defined as airway hyper-responsiveness causing variable and reversible airflow obstruction

Triggers of airway hyper-responsiveness include:

Upper respiratory tract infections (URTIs)

Allergens (pollen, animal dander, dust, mold, etc)

Air pollution, cigarette smoke, other chemicals

Drugs (aspirin, NSAIDs, Beta-blockers)

Cold air

Exercise

Guide For Pathophysiology

Sue E. Huether, Kathryn L. McCance



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