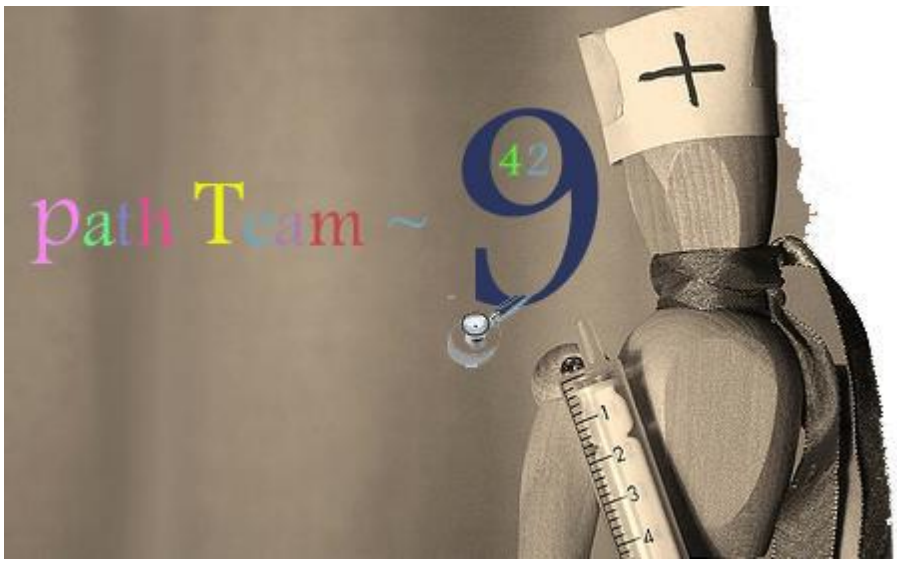

Organized by:
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❖ What triggers an attack of asthma?

a) Extrinsic, or allergic, asthma is triggered by environmental allergens, including dusts, pollens, dander, foods, etc.

b) The most common provocative agents of intrinsic (nonimmune) or idiosyncratic asthma are infections of the respiratory tract, especially viral.

- Other types of intrinsic asthma include drug-induced asthma (many drugs, especially aspirin); occupational asthma, provoked by fumes, organics, chemicals; and exercise-, cold-, and emotional stress-induced asthma.

❖ What is the mechanism of the early symptoms, and what are the causes of effects that appear several hours after exposure to an allergen?

- Acute early-phase reaction:
  - Exposure of presensitized IgE-coated mast cells to the same or cross-reacting antigens results in an acute early-phase reaction from the release of:
  - histamine (which causes bronchospasm)
  - leukotrienes (which attract leukocytes and eosinophils and release mucus)
  - platelet activating factor (which causes more release of histamine and serotonin from platelets).
- Late-phase effects:
  - Cytokines released by leukocytes, eosinophils, and basophils recruited during the early phase contribute to the late-phase effects.
  - Histamine from basophils causes bronchoconstriction and edema
  - neutrophils cause inflammatory damage;
  - and the major basic protein of eosinophils causes epithelial damage and shedding and contributes to bronchoconstriction.

❖ Are all forms of asthma associated with type I hypersensitivity reactions?

- No. Intrinsic asthma is not triggered by type I hypersensitivity. The precise causes of hyperreactive airways in intrinsic asthma are not known.

❖ **What is responsible for the accumulation of eosinophils?**

- Eosinophils are attracted by chemotactic factors released by mast cells and also by the chemokine eotaxin. The latter is produced by bronchial epithelial cells. Eosinophil accumulation is also favored by IL-5, a T-cell-derived cytokine.

❖ **What role do eosinophils play in causing tissue damage?**

- Major basic protein of eosinophils causes epithelial damage and shedding.

❖ **Are mucous plugs diagnostic of bronchial asthma?**

- No

❖ **What are the causes of bronchiectasis?**

➢ The most common conditions are:

a) Bronchial obstruction (tumors; foreign bodies), diffuse obstructive airway diseases (atopic asthma; chronic bronchitis)
b) Congenital or hereditary conditions (congenital bronchiectasis; cystic fibrosis)
c) Necrotizing or suppurative pneumonias.

✓ Those conditions predispose to chronic necrotizing infections and consequent destruction of muscle and elastic support tissue of bronchi and bronchioles.

❖ **Why does it seem to affect the lower lobes preferentially?**

- Inhaled materials and secretions tend to gravitate to the lower lobes.

❖ **What type of inflammatory cells might you expect? Compare with asthma.**

- Acute inflammatory cells, especially PMNs, are present, although evidence of chronic inflammation is also likely to be present.
- By comparison, in asthma, the inflammatory cells are composed of CD4+ lymphocytes, eosinophils, mast cells, and a few neutrophils.

❖ **What is the definition of asthmatic bronchitis?**

- Some patients with a clinical definition of chronic bronchitis have hyperresponsive airways with intermittent bronchospasm.

Summary

Asthma: Dyspnea and wheezing

Types	• 1. Extrinsic asthma: Type 1 Hypersensitivity reaction, IgE, childhood, family Hx of allergy. • 2. Intrinsic asthma: associated e bronchial asthma, aspirin, exercise, cold induced. No Hx of allergy
Morphology	•Hypertrophy of bronchial smooth muscle & hyperplasia of goblet cells e eosinophils •Mucous plug e Curschmann spirals & Charcot-Leyden crystals.
Complication	•Superimposed infection •Chronic bronchitis •Pulmonary emphysema •Status asthmaticus

Bronchiectasis: Chronic necrotizing infection of the bronchi and bronchioles leading to permenant dilatation of these airways

Causes	• Infection • Obstruction • Congenital (Cystic fibrosis, Kartagener's Syndrome)
Clinical features	• Sever persistent cough with sputum (mucopurulent sputum) sometime with blood. • Clubbing of fingers.
complications	• If sever, obstructive pulmonary function develop. • Lung Abscess • Rare complications: metastatic brain abscess and amyloidosis.