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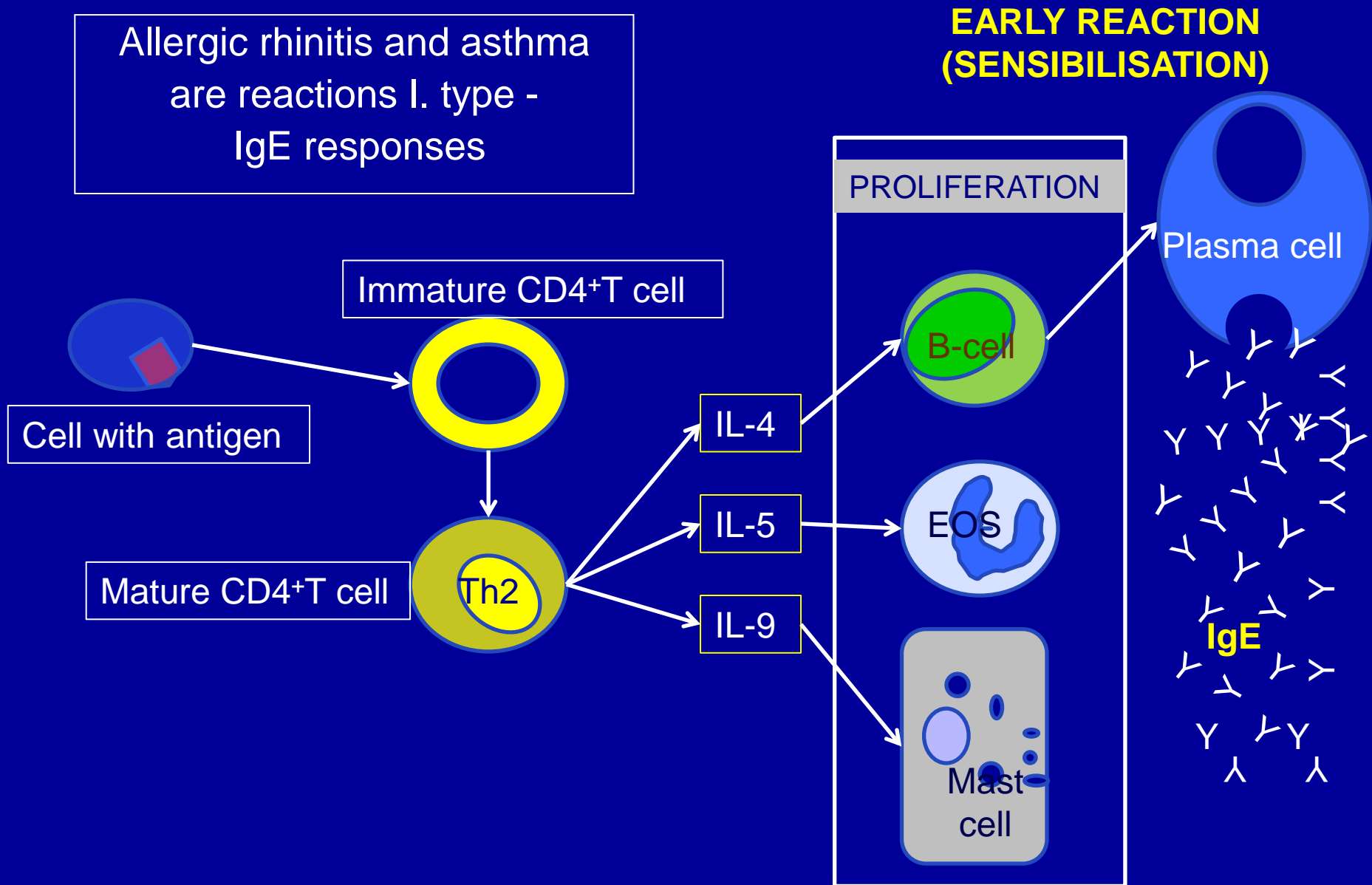


Occupational allergic diseases

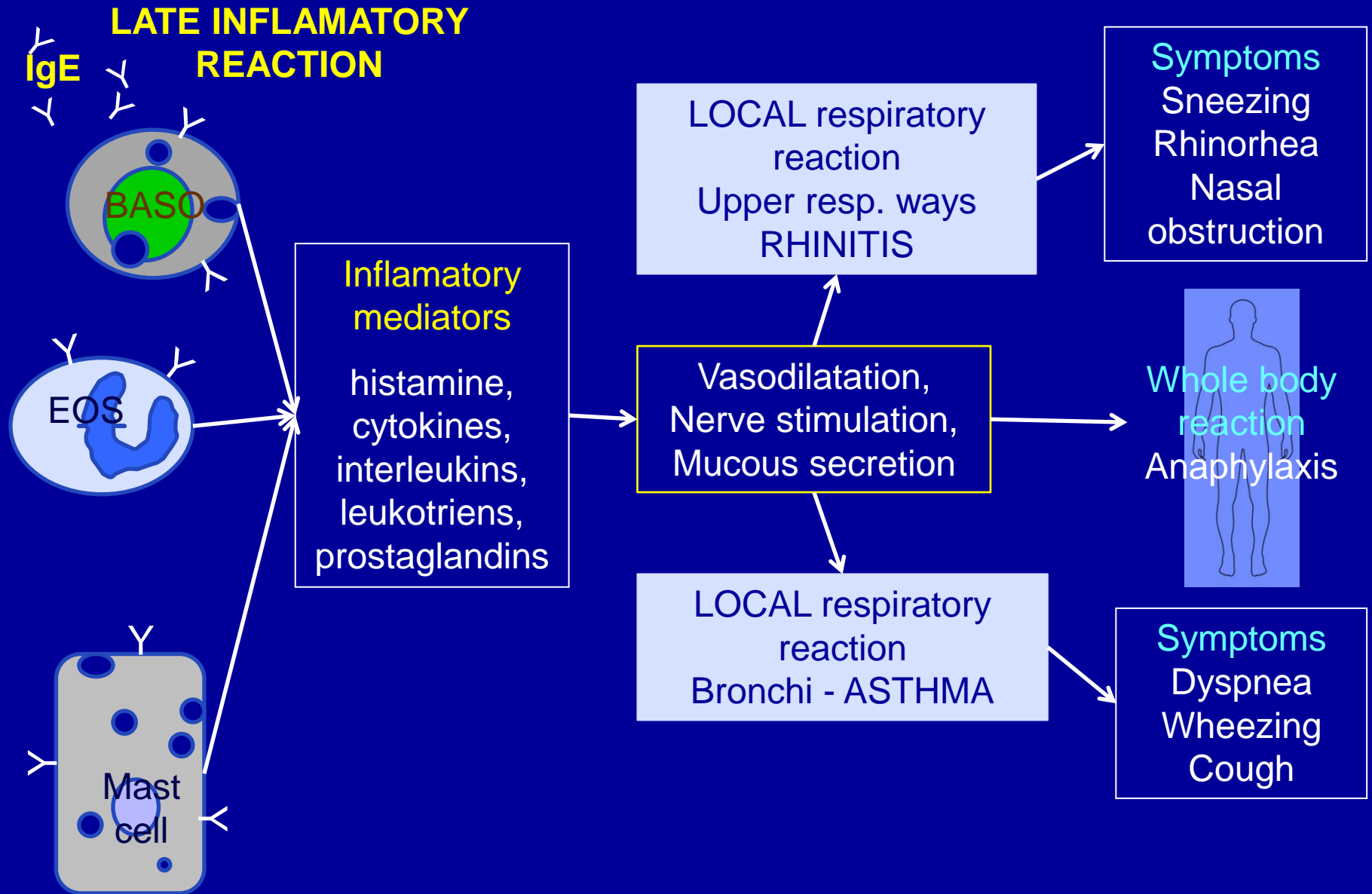
Occupational allergic rhinitis
Occupational asthma
Hypersensitivity pneumonitis

Occupational allergic diseases

Allergic rhinitis and asthma
are reactions I. type -
IgE responses



Occupational allergic diseases



Occupational allergic diseases

- Etiology – contact with allergens
 - Animal products
 - Avian proteins, feathers
 - Excreta – rat, mouse, guinea pig, other laboratory animals
 - Thermophilic actinomycetes
 - Bee venom
 - Vegetable products
 - Flour dust
 - Castor bean
 - Colophony
 - Green coffee dust
 - Chemicals
 - Isocyanates, platinum salts, trimellitic anhydride,
 - Dust from epoxides, nickel, formaldehyde, ammonia, chlorine, PVC

Professional allergic rhinitis

■ Clinical features

- **Allergic rhinitis** – sneeze, hypersecretion, nose congestion
 - Symptoms started immediately after contact with allergen or late from 8 – 24 hours
- **Irritative rhinitis** – erythema of nasal mucosa, nose congestion and hypersecretion
 - Extreme symptoms – perforation of nasal septum (chromic acid, hydrofluoric acid)

■ Diagnostic

- Occupational history, Clinical features
- Special examinations – Rhinoendoscopy, rhinomanometry
 - Cytology – Allergic rhinitis – Eosinophils, Mast cells, Neutrophils
 - Irritative rhinitis – Neutrophils – no other
 - **Specific IgE,**
 - Provocation test,

■ Treatment

- Exclusion from exposure, Antihistamines, Local corticoids, local decongestantes (Nasivin)

Professional allergic asthma

■ Etiology

- Allergic form – with immunologic reaction
- Irritant (non-allergic) form - without immunologic reaction – induced by chemical and irritative factors

Plant allergens	Flour dust, grain dust Pollen of trees, grasses, flowers Flax, cotton, hempstead, dust from wood
Animal allergens	Leather, fur, hairs and other biological material of domestic and farm animals Feathers, birds and poultry secretions Insects, mites, oysters, crabs
Chemicals	Isocyanates, Metals and their salts – Ni, Cr, Co ... Insecticides – organophosphate, pyrethroids Medicaments – penicillins, cephalosporins isoniazid Formaldehyde, ethyleenoxide

Professional allergic asthma

- Clinical picture
 - Asthma attack
 - Asymptomatic stage
- According to clinical picture – mild, medium, heavy form
- Symptoms - dyspnea, wheezing, and/or cough that correlate with workplace exposures
- Patients often report feeling better in the evenings or during weekends and vacations

	No occupational asthma	Occupational asthma
Dyspnea	During the night or early morning	During the stay in work
Other workers	Don't have similar symptoms	Have a similar symptoms
Work environment	Without allergens or irritant	Allergens and irritant substance

Professional allergic asthma

■ Diagnostic

- Medical history
 - Family and personal
 - Occupational history
 - when started the health problems,
 - when is the patient without problems,
 - with what he is working ...
- Physical examination – can be normal
- Functional examination of lungs – obstruction, bronchial, hyperreactivity
- Bronchomotoric nonspecific test – after inhalation of acetylcholine or histamine – test is positive when the FEV₁ decrease more than 20%
- Re-exposure test – symptoms occurs after return to work

■ Treatment

- β_2 adrenergics – terbutalin, salbutamol, fonoterol
- Anticholinergics, corticoids
- Exclusion from exposure

Hypersensitivity pneumonitis = Extrinsic allergic alveolitis (EAA)

- EAA – is immunologically mediated inflammatory disease of the lung parenchyma that is induced by inhalation of organic dusts that contain a variety of etiologic agents
- Essential of diagnosis
 - A link between symptoms and antigen exposure in the work
 - The antigen can be
 - microbial agent
 - animal protein
 - chemical sensitizer
 - The clinical presentation can be
 - acute, subacute, chronic

Hypersensitivity pneumonitis = Extrinsic allergic alveolitis (EAA)

Antigen		Exposure	Syndrome
Bacteria	Thermoactinomyces vulgaris	Moldy grain, compost	Grain worker's lung
	Thermoactinomyces sacchari	Moldy sugar cane fibers	Bagasosis
	Faenia rectivirgula	Moldy hay	Farmer's lung
Fungi	Aspergillus clavatus	Moldy malt	Malt worker's lung
	Penicillium casei	Moldy cheese	Cheese worker's lung
Animal proteins	Avian proteins	Feathers, bird droppings	Bird breeders's lung
	Rodent proteins	Sera, urine, pelts	Animal handler's lung
Chemicals	Diisocyanates	Paints, polyurethane foam	Isocyanate lung

Hypersensitivity pneumonitis = Extrinsic allergic alveolitis (EAA)

- Hypersensitivity pneumonitis is initially characterized by alveolitis and granulomatous pneumonitis.
- Continued antigen exposure may lead to progressive interstitial fibrosis.
- Inhalation exposure to any antigen in a sensitized person may result in **acute or chronic** presentations of hypersensitivity pneumonitis depending on the exposure conditions
- Acute form of presentation of hypersensitivity pneumonitis usually occurs from 4 to 6 hours of the end of intense exposure antigen

Hypersensitivity pneumonitis = Extrinsic allergic alveolitis (EAA)

Clinical findings in case of acute form:

- Symptoms: chills, fever, malaise, myalgia, cough, headache, dyspnea
- Physical examination: Bibasilar inspiratory crackles on chest
- EAA is often misdiagnosed as an acute viral syndrome or pneumonia
- Laboratory findings: Peripheral blood leukocytosis with neutrophilia and a relative lymphopenia.
Arterial blood gas values may show hypoxemia.
- Chest radiographic findings
 - May be completely normal even in symptomatic individuals
 - Acute phase is associated with the presence of a reticulonodular pattern
 - Infiltrates are usually bilaterally distributed

Hypersensitivity pneumonitis = Extrinsic allergic alveolitis (EAA)

Clinical findings in case of progressive form:

- Physical examination: cyanosis and inspiratory crackles
- Pulmonary function tests:
 - Decrease in the FEV1 and FVC
 - Restrictive respiratory disorder
- Chest radiographic findings
 - X- ray – Diffuse linear opacities
 - HRCT
 - Centrilobular micronodules
 - Ground-glass opacities
 - Linear shadows

Hypersensitivity pneumonitis = Extrinsic allergic alveolitis (EAA)

- **Complications** - development of irreversible lung fibrosis
- **Prevention** - Respiratory protective equipment, complete removal of the worker from exposure is necessary.
- **Treatment**
 - Progressive EAA – Corticosteroids (Prednisone 1 mg/kg/d)
 - Bronchospasm - beta-agonists
 - Supplemental oxygen - patients with hypoxemia
 - Lung transplantation is recommend in cases of progressive end-stage illness.