OCCUPATIONAL RESPIRATORY DISEASES

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- The respiratory tract is often the site of injury from occupational exposures
- The respiratory tract has a limited number of ways to respond to injury
- Acute : rhinosinusitis, laryngitis, …
- Chronic : asthma, parenchymal fibrosis, …

Evaluation of Patients

- Detailed history (occupational & environmental exposures, hobbies)
- Thorough physical exam (not specific, insensitive)
- Appropriate imaging studies
- Pulmonary function tests (spirometry, bronchoprovocative tests)

Types of Diseases

- Toxic inhalation injury
- Occupational asthma
- Hypersensitivity pneumonitis
- Inhalation fever
- Pneumoconioses
- Chronic bronchitis
- Pleural disorders
- Lung cancer

Toxic Inhalation Injury

- Short term exposure to high concentrations of noxious gases, fumes, mists
- Severity & site of injury depends on:
  - Water solubility
  - Concentration
  - Duration of exposure
  - Ventilation of victim

Treatment

- Decontamination
- O2
- Bronchodilator
- Drainage
- No steroids unless the patient is not recovering promptly

Asthma

- Reversible, episodic airway obstruction (spontaneously or with treatment)
- Airway inflammation
- Increased airway responsiveness

- Occupational asthma is the most common occupational respiratory disorder

Work-related Asthma

- Occupational asthma (3%)
- Work-aggravated asthma

- Work-related asthma(15%)

Occupational Asthma

- Sensitizer-induced
  - long term exposure
  - specific responsiveness to an etiologic agent
- Irritant-induced (RADS)
  - a single intense exposure
  - persistent non-specific hyperresponsiveness

- Non-specific hyper-responsiveness is a very important characteristic of allergic & non-allergic induced asthma

Sensitizing Agents

- HMW agents (IgE-mediated)

  e.g. animal & plant proteins

- LMW agents (IgE-mediated , unknown)

  e.g. antibiotics , metals

  acid anhydrides , diisocyanates

Non-sensitizing Agents

- Anticholinesterase effect

  e.g. organophosphates

- Endotoxin effect

  e.g. cotton dust

- Airway inflammation

  e.g. acids, ammonia, chlorine

- Airway irritation

  e.g. dusts, fumes, mists, vapors, cold

Sensitizer-induced Asthma

- Early response (HMW) (15-30 min)
- Late response (LMW) (4-8 hours later)
- Dual response (HMW)

Diagnosis

❶ Confirming the diagnosis of asthma

- intermittent respiratory symptoms
- evidence of reversible airway obstruction

❷ Relationship between asthma & the work environment

- symptoms occur only at work
- symptoms improve on weekends or vacations
- symptoms occur regularly after work shift
- symptoms progressively increase over the course of the work week
- symptoms improve after a change in the work environment

❸ At least one of the symptoms (wheezing, shortness of breath, cough, chest tightness) should occur while the worker is at or within 4-8 hours of leaving the workplace

Note : With persistent exposure symptoms become chronic & lose relationship to work

Spirometry

- The most reliable method
- May be normal between attacks
- After bronchodilator : 12% increase in FEV1 is significant (hyper- responsiveness)
- Across work shift : 10% fall in FEV1 is suggestive

PFM

- PEFR is the best way
- 4 times/day + symptoms + medications
- ≥ 20% diurnal variability is considered asthmatic response

Prevention

- In all work places where cases are diagnosed
- Removal from exposure
- Engineering controls
- Medical surveillance
- Education
- Smoking cessation program

Hypersensitivity Pneumonitis
Extrinsic Allergic Alveolitis

- Immunologically-mediated inflammatory disease of lung parenchyma
- Induced by inhalation of organic dusts containig the etiologic agents

Causing Agents

- Bacteria (Thermoactinomycetes in moldy hay, grain, sugar cane,…)
- Fungi (Aspergillus in moldy malt, cheese,…)
- Amoeba (Acanthamoeba in contaminated water)
- Animal proteins (bird feathers, urine, droppings,…)
- Chemicals (toluene diisocyanate, TMA, … in polyurethane foams, epoxy resins,…)

Pathogenesis

- Repeated inhalation exposure
- Sensitization of the individual
- Immunologically-mediated lung damage

Course

- Few exposed people develop it
- Complete resolution in early removal
- Progressive interstitial fibrosis in continued exposure

Acute HP

- More common
- 4-6 hours of an intense exposure
- Chills & fever
- Malaise & myalgia
- Dyspnea & cough
- Headache
- Bibasilar inspiratory crackles

LAB

- Diff. : Leukocytosis (increased neutrophils, decreased lymphocytes)
- ABG : hypoxemia
- CXR : bilateral reticulonodular pattern
- Spirometry : restrictive

Course

- Progresses 18-24 hours, then gradually resolves
- Recurrence occurs with re-exposure

Chronic HP

- Recurrent low-level exposure
- Insidious onset
- Chronic interstitial lung disease with fibrosis

Symptoms & Signs

- Dyspnea & cough
- Excessive fatigue
- Weight loss
- Cyanosis & clubbing
- Inspiratory crackles

CXR & Spirometry

- Diffuse linear markings
- Reduced lung size

- Spirometry : restrictive

Treatment

- Avoidance of exposure
- Respiratory protective equipment
- Corticosteroids (Prednisone 1mg/kg/day – 1 month) – follow with CXR & PFT
- Beta-agonists
- Oxygen

Inhalation Fever

- Exposure to polymer fumes, metal fumes, organic dusts (grain, cotton,…)
- High attack rate
- Most common cause : zinc oxide
- Chills, fever, myalgia, headache, malaise, cough, chest discomfort
- CXR, PFT, ABG : normal
- CBC : leukocytosis
- Treatment : symptomatic
- Complete resolution in 1-2 days
- No evidence for steroid therapy

Pneumoconioses

- Silicosis
- Asbestosis
- Coal workers' pneumoconiosis

Pneumoconiosis

- Literally means dust in lungs
- Not all dusts cause disease
- ILO : accumulation of dust in lungs and the tissue reaction to its presence

Benign Pneumoconiosis

- Inert dusts contain < 1% quartz
- Do not disrupt alveolar architecture or give rise to collagenous fibrosis
- No symptoms or functional abnormalities
- May be cleared form lungs over a period of time with avoidance of exposure
- e.g. iron, tin, antimony, barium

Collagenous Pneumoconiosis

- Dusts that stimulate a response in lungs and lead to irreversible fibrosis and structural alterations
- e.g. silicosis, asbestosis

Chronic Bronchitis

- Minerals (coal, silica, cement,…)
- Metals (vanadium, welding fumes,…)
- Organic dusts (cotton, grain,…)
- Smoke (tobacco, fire,…)
- Esp. in smokers
- Increased incidence among coworkers

Pleural Disorders

- Benign pleural effusions
- Pleural plaques
- Mesothelioma
- The primary cause is asbestos
- Not always accompanied by asbestosis

Lung Cancer

- Asbestos
- Arsenic
- Chloromethyl ethers
- Chromium (hexavalent)
- ETS (synergism)
- Mustard gas
- Nickel
- PAH
- Radon
- Silica
- Beryllium

- ***GOOD LUCK***