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Occupational lung disease

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Occupational lung disease

- Respiratory tract is often the site of injury from occupational exposure.
- The widespread use of potentially toxic material in the workplace pose a major threat to both airways and parenchyma.

Evaluation of patients with occupational lung disease

1. Detailed history
2. Physical exam
3. Imaging studies
4. Pulmonary function test

history

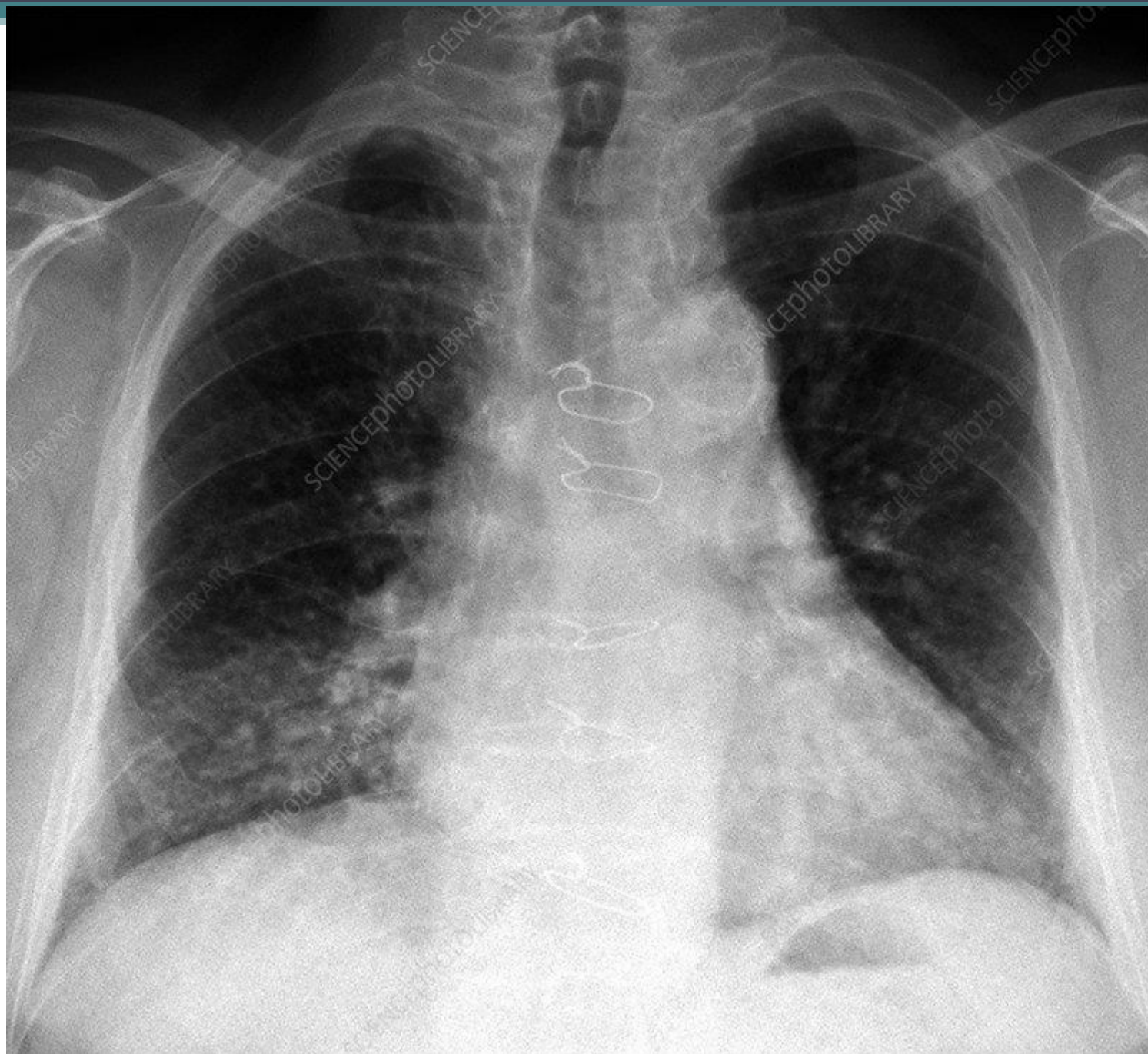
- Occupational & enviromental exposure
- Duration and type of exposures
- Substance data sheet(SDS) or MSDS
- Patients home,hobbies,social habits

Physical exam

- Helpful but insensitive
- Do not present with special clinical finding
- Cyanosis,clubbing
- irritation of oropharyngeal & nasal
- Presence of wheezing , rhonchi , crackle
- Isolated right ventricular failure

Imaging studies

- Normal CXR do not exclude damage
- Abnormal CXR can be seen without lung injury.
- Abnormality on CXR do not necessarily correlate with degree of pulmonary impairment (better assessed by PFT , ABG)
- CT is better to detect abnormality of pleura & mediastinal structures
- HRCT for assessing severity emphysema & ILD



Pulmonary function test

- PFT is used to detect & quantify abnormal lung function.
 - Include: measurement of lung volume, diffusing capacity, gas exchange ,exercise test.
- ✓ Performance requirement; ATS & ERS

SPIROMETRY

- Best method of detecting the presence and severity of airway obstruction.
- Most reliable assessment of overall respiratory impairment.
- The results can be predicted values from reference populations (adjust for age, height, sex)
- 10-15% lowering the predicted value for smaller lung of nonwhites.
- NIOSH produce separate reference-value for whites, african-americans ,...

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Peak expiratory flow rate(PEFR)

- Single-breath test
Reflects the degree of airway obstruction
- ✓ Self recording
- ✓ Specially valuable in the diagnosis of asthma(delayed responses after the shift work)
- ✓ Limitation ;malingering

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BRONCHOPROVOCATION TESTS

- Useful in the diagnosis of asthma
- Pulmonary Function response to histamine and methacholine
- Serial Fev1 obtained repeatedly after increasing doses of histamine or methacholine.
- Specific inhalation challenge test.

INTERPRETATION OF TEST RESULTS

• PC 20.Mg/ml	DEGREE OF BHR
1. >16mg	Normal
2. 4-16mg	borderline
3. 1-4mg	Mild BHR (+)
4. <1mg	Moderate to severe BHR

Modified from Crapo RO, Casabian R, Cortes AL, et al: Guidelines for methacholine and exercise challenge testing—1999. This official statement of the American Thoracic Society was adopted by the ATS Board of Directors, July 1999. *Am J Respir Crit Care Med* 161:309–329, 2000.

Site of respiratory tract deposition and effects

High	Ammonia, formaldehyde	Upper airway
Moderate	Chlorine, sulfur dioxide	Lower airways
Low	Nitrogen oxides, phosgene	Lung parenchyma
Particle Size (Aerodynamic Diameter)		
>10 μm	Dust from Earth's crust	Upper airway
2.5-6 μm	Some fire smoke particles	Lower airways
<2.5 μm	Metal fumes, asbestos fibers	Lung parenchyma

TOXIC INHALATION INJURY

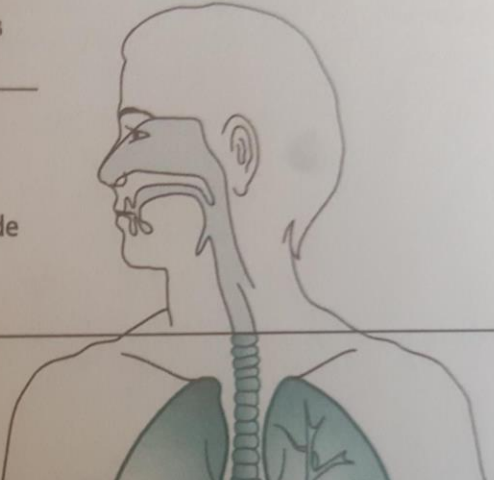
Short term exposure of high concentration of noxious **gases , fumes , mist.**

Generally are result of industrial or transportation accident or fire.

Effects can range from mild conjunctival and upper respiratory irritation in low-dose exposure to life-threatening laryngeal or pulmonary edema in high-dose exposure

- The site of deposition of an inhaled gas is determined primarily by water solubility.
- Other important factors are the duration of exposure and the minute ventilation.

Water solubility	Initial level of impact	Compounds
High	Eyes	Aldehydes
	Nose	Ammonia
	Pharynx	Sulfur dioxide
	Larynx	
Medium	Trachea	Chlorine
	Bronchi	



Water solubility

soluble(Amonia,chlorine)

Mild exposure;

Local irritation of conjunctival membrane and upper airway

Moderate exposure;

hoarsness, cough ,bronchospasm

Acute high level;

ARDS

Phosgene,oxides ofnitrogen

- Mild irritation of upper respiratory tract.
- Deposit in lower respiratory tract causes irritation in parenchyma and tissue necrosis

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Site of Injury	Acute Effects	Chronic Effects
Eye, nose, sinuses, oropharynx Upper airway Lower airways	Irritation, inflammation Laryngeal edema, upper airway obstruction Tracheobronchitis, bronchorrhea, decreased mucociliary clearance	Corneal scarring, nasal polyps Laryngeal polyps Asthma, bronchiectasis
Lung parenchyma	Pneumonitis, pulmonary edema/adult respiratory distress syndrome	Pulmonary fibrosis, bronchiolitis obliterans

Heavy exposure

- 1-If Nose and throat are badly **burned** or if there is **Hoarsness** or **stridor** chemical laryngitis should be suspected.
- 2-The presence of **early wheezing** suggest that the exposure was relatively heavy.
- 3-CXR usually will be normal immediately after exposure,ARDS may develop 4-8 hours of heavy exposure.
- 4-ABG may show **hypoxemia before radiographic evidence** of parenchymal injury.

5-In **poorly water-soluble agents** such phosgene and oxides of nitrogen ,patient should be observed for a minimum of 24 hours.

Hydrofluride toxic inhalation injury



Occupational asthma

Characterized by reversible
airway obstruction ,
(either spontaneously or with
treatment) airway inflammation and
increased responsiveness to a variety
of stimuli.

- More than 250 agents in the workplace cause asthma ,and the list is growing as new materials are introduced.

Work-aggravated asthma

- When workplace exposures lead to exacerbation of preexisting nonnoccupational asthma

Reactive airway dysfunction syndrome (RADS)

A term used to describe irritant-induced asthma, caused by a short term ,high intensity exposure.

Asthma

- 1) **Sensitizer-induced asthma**
- 2) **Irritant-induced asthma**

Sensitizing agents to cause asthma

High-molecular weight compound

- >1000 Da
- Type I(IgE)-mediated reactions
- Specific responsiveness to the etiologic agent

Low-molecular-weight compound

- <1000Da
- Unknown mechanism
- Persistent nonspecific airway hyperresponsiveness (but not specific)

Table 20-3. Some agents causing occupational asthma.

Mechanism	Examples
Without "sensitization" Anticholinesterase effect Endotoxin effects Airway inflammation Airway irritation	Organophosphate pesticide (agricultural workers) Cotton dust (textile workers) Acids, ammonia, chlorine (custodial workers, paper manufacturing workers) Dusts, fumes, mists, vapors, cold (construction workers, chemical workers)
With "sensitization" High-molecular-weight agents IgE-mediated (complete allergens) Low-molecular-weight agents IgE-mediated (haptens) Mechanism undefined	Animal and plant proteins (laboratory workers, bakers) Antibiotics, metals (pharmaceutical workers, metal plating workers) Acid anhydrides, diisocyanates, plicatic acid (epoxy plastics and paints, polyurethane foams and paints, western red cedar products)

pathogenesis

- Asthmatic air way are characterized by;
1-infiltration with inflammatory cells(eosinophils)
2-edema
3-loss of epithelial integrity

Inhalation of specific antigen in sensitizer-induced asthma will trigger;

I. rapid-onset ,Self-limited bronchoconstriction called **Early response**.

II. In many sensitized workers a delayed reaction will occur 4-8 hours later called **late response** (LMW)

Persistent hyperresponsiveness, inflammation, obstruction

III. Some workers **dual response**, and in other isolated late response. (HMW)

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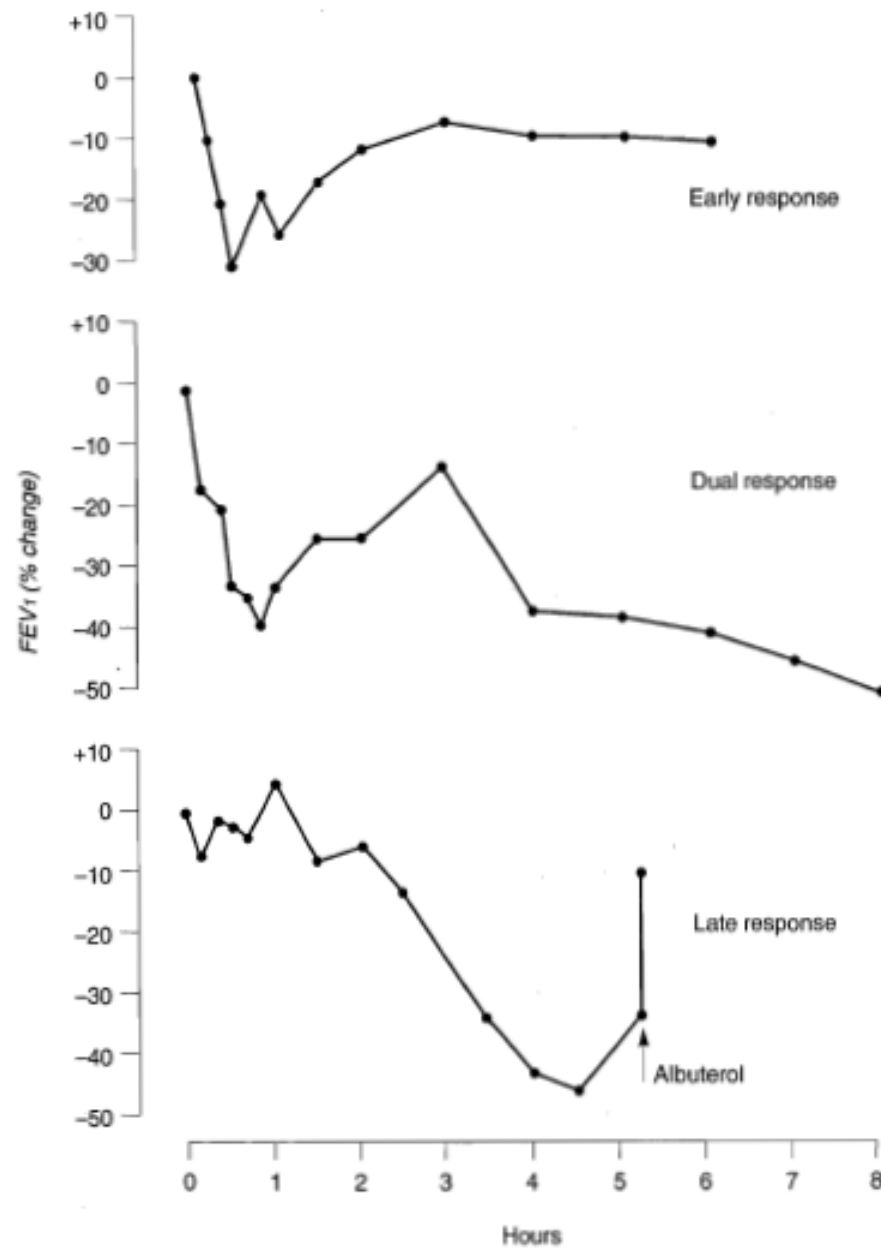


Figure 20-1. Potential responses to inhalation of allergen in sensitized workers with asthma.

Irritant-induced asthma;

- Mechanism of inflammation are not well understood.
- The axonal reflex involving C-fiber and release of neuropeptides.

Other mechanism of irritant-induced

- Reflex bronchoconstriction ; stimulation of neuroreceptors by;
cold air , dusts , mists , vapors , fumes .
Does not lead to inflammation .

usually in work-aggravated asthma .

Other mechanism of irritant-induced asthma

- Pharmacologic bronchoconstriction ;
an agent in the workplace causes the direct release of mediators ;

Cotton dust in textile mills

direct effect on the autonomic regulation ;
organophosphate pesticides

Diagnosis

- Intermittent respiratory symptoms and evidence of reversible or variable airway obstruction are present.
- The diagnosis of occupational asthma is made by;
 - ✓ Confirm the diagnosis of asthma
 - ✓ Establishment between asthma and work environment

Relationship between asthma and workplace exposure

1. Symptoms occur **only at work**
2. Symptoms improve **on weekends** and vacation
3. Symptoms occur regularly **after the work shift**
4. Symptoms increase progressively over the course of the workweek.
5. Symptoms improve after a change in the work environment.

At least one of the symptoms of;

- Cough
- wheezing
- Chest tightness
- Shortness of breath
- ✓ at work or within 4-8 hours of leaving workplace
- ✓ worker's symptoms improve during days off.
- ✓ Persistent exposure tend to lose relationship.

HMW sensitizers cause early or dual response while LMW sensitizers tend to induce isolated late response that may occur hours after the work shift is over.

Evaluation

- Concomitant eye & upper respiratory symptoms
- Recurrent episodes of bronchitis
- Presence of symptoms in other workers
- Physical exam
- CXR

spirometry is the most reliable method for assessing airway obstruction.

Because asthmatic patients typically have reversible airway obstruction, they have normal lung function between acute attacks.



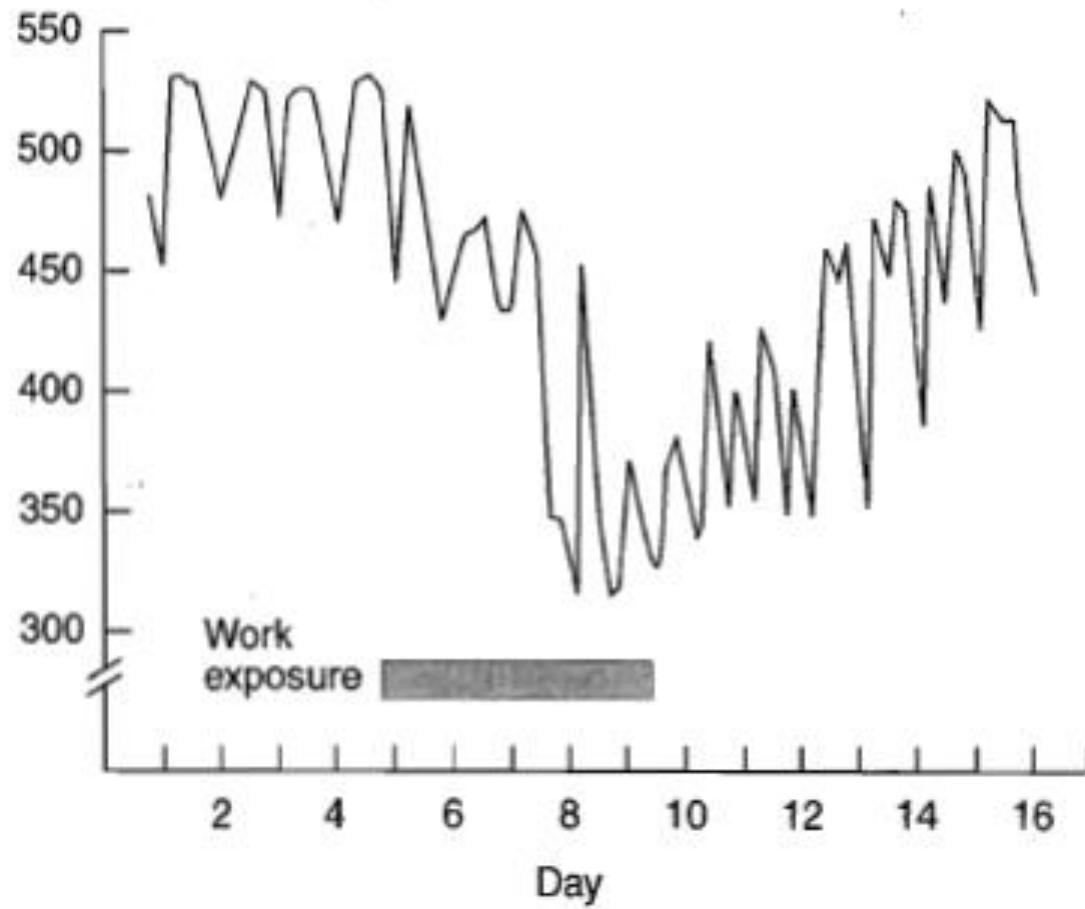
Response to bronchodilator administration

- A 12% improvement in FEV1 of at least 200ml after bronchodilator is how ATS defines a significant improvement, indicative of hyperresponsiveness airways.
- **Across-shift spirometry**; more than 10% fall in FEV1 is suggestive of an asthmatic response.

PEFR

- AT Least 4 time while awake.
- A 20% or greater diurnal variability in PEFR considered evidence of an asthmatic response.

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MCT & HCT

- Suspected of having occupational asthma in workers with normal spirometry.
- Valuable if it demonstrates an increase in airway responsiveness on returning to work or decrease when away from work.

Allergy skin test

- The worker is atopic or not?
- Atopy is a risk factor for HMW ASTHMA.
- Skin test with flour , animal proteins , coffee.
- Skin test may be helpful for a few LMW compounds like platinum salts.
- RAST or ELISA may confirm exposure to allergens like flour, animal proteins ,acid anhydride , plicatic acid, isosyanates.

treatment

- Reduce or eliminate the worker's exposure.
- Modification in the workplace.
- Substitute the offending agent with safer one.
- Improved local exhaust ventilation.
- Personal protective equipments(irritant-induced asthma)
- Follow-up visit
- In sensitizer-induced may be necessary to remove from the workplace.
- Avoidances from other irritant(dust,mist,smoke..)

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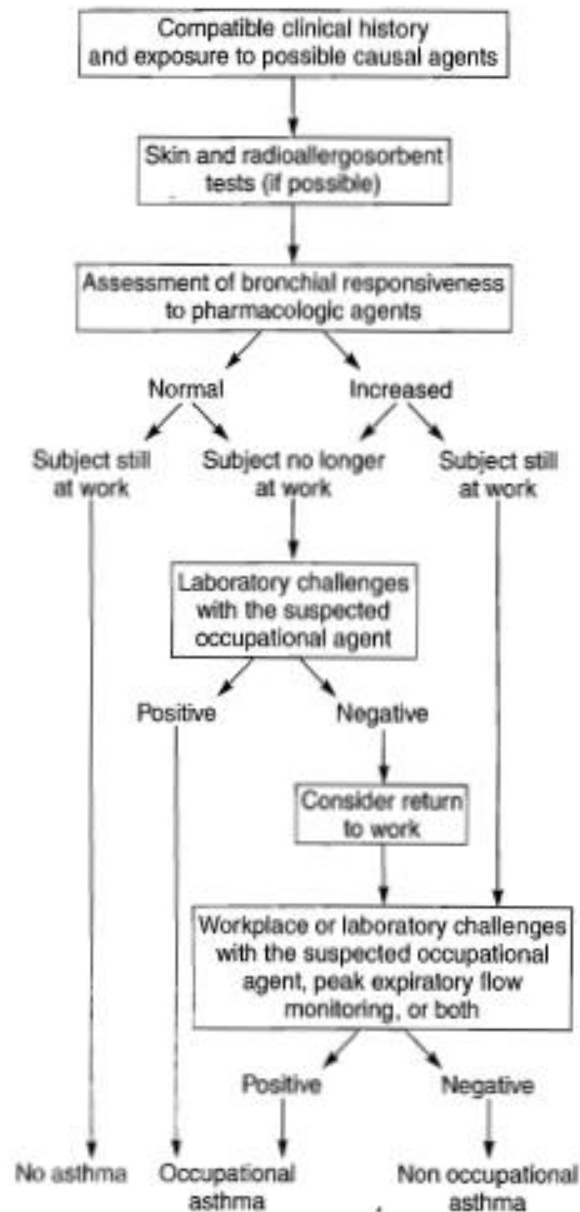


Figure 20-4. Algorithm for the clinical investigation of occupational asthma.

Inhalation fevers

Inhalation fever refer to several syndromes that are characterized by short-term but debilitating flulike symptoms after exposure to organic dusts, polymere fumes, metal fumes

In contrast to asthma, which require susceptibility and/or sensitization, the attack rate is high.

- ✓ Metal fume fever
- ✓ Polymer fume fever
- ✓ Organic dust toxic syndrome

Metal Fume Fever

- Inhalation of certain freshly formed metal oxides can cause metal fume fever .
- An acute self-limiting flulike syndrome.
- The most common cause of this syndrome is the inhalation of Zinc oxide, generated from molten bronze or welding galvanized steel.
- Other metal include copper , magnesium.

- When zinc heated to melting point zinc oxide fumes are generated.
- 0.1 - 1 micrometer
- Leukocytes recruitment to lung and release of cytokines.

Clinical finding

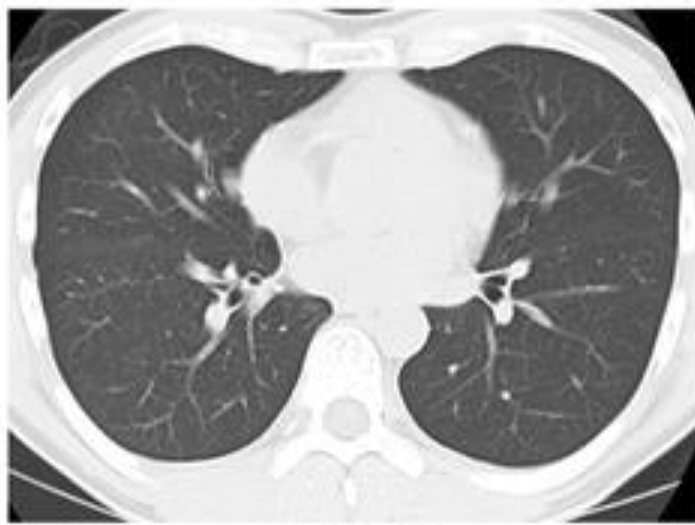
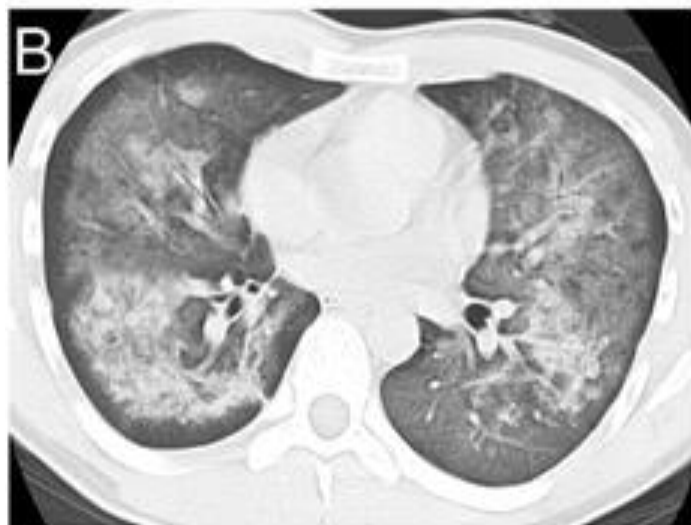
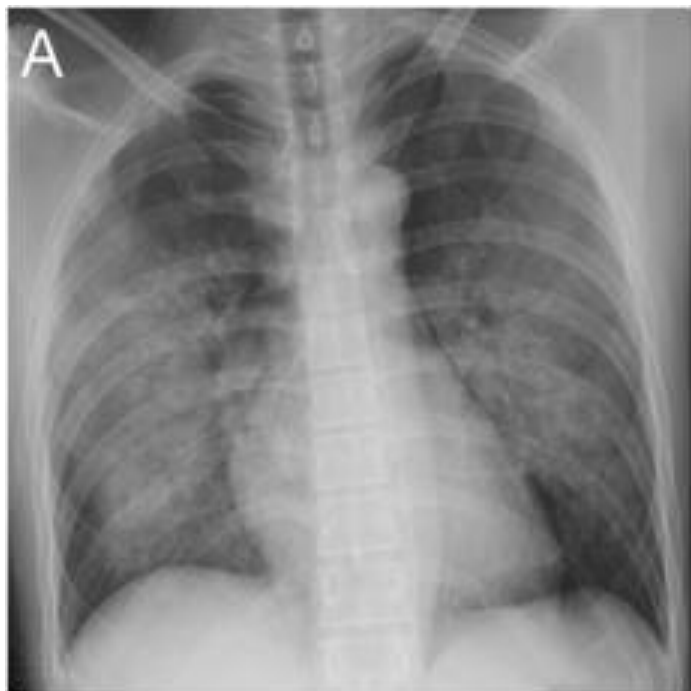
- Clinical syndrome begin 3-10 hours after exposure to zinc oxide.
- Initial symptoms: metallic taste, throat irritation, fever, chills, myalgia, malaise, nonproductive cough, occasionally nausea, vomiting, headache.
- Physical exam; crackles
- Lab; leukocytosis (left shift), LDH elevated
- CXR, PFT, ABG are usually normal
- Signs & symptoms peak at 18 hours, resolve 1-2 days

Polymer Fume Fever

Combustion products of polytetrafluoroethylene

- when teflon temperatures greater than 300/c
- Welding of metal coated with teflon.
- Cigarettes
- Fever to develop within several hours.
- Sign&symptoms, Lab are the same as MFF
- The syndrome is self-limiting, resolves 12-48hours
- Exposure to very high concentrations tend to severe chemical pneumonitis and pulmonary edema.

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On admission

Day 9

Organic Dust Toxic Syndrome

- Inhalation of various bioaerosols contaminated with fungi , bacteria , endotoxins can cause an acute febrile syndrome.
- Moldy silage , moldy wood chips,compost ,grain dust ,cotton dust(mill fever),..

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