RUTGERS
School of Health Related Professions

Pulmonology

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Part I:
Infectious Disorders

- Influenza
- Acute Bronchitis
- Pneumonia
- Tuberculosis
- Epiglottitis
- Pertussis

Influenza

Background:

- occurs as epidemics or pandemics (type A)
- most frequently in fall / winter

Etiology:

- orthomyxovirus
- three antigenic subtypes
  - A & B (A & B are similar clinically)
  - C (milder)
- transmitted via large resp droplets; incubation 1-4 days
- Type A further divided into subtypes based on:
  - hemagglutinin (H) with 18 subtypes
  - neuraminidase (N) with 10 subtypes

Influenza

Clinical Findings:

- abrupt onset, fever, chills, headache, coryza, myalgias (esp. back and legs), sore throat, proteinuria, leukopenia, cervical lymphadenopathy
- epithelial necrosis leading to bacterial superinfection (esp. with pneumococcus or *S. aureus)*

Diagnosis:

- usually clinical (aka presumptive)
- rapid Ag tests (nasal/pharyngeal)
- fever and cough in areas of epidemic:

  positive predictive value of 80%

Influenza

Comparison of Influenza treatments			
	Prophylaxis	**Treatment (w/i 48 hrs)**	**Ages**
amantadine	**A**	**not recommended**	**≥ 1**
rimantadine	**A**	**not recommended**	**≥ 1**
oseltamivir PO (Tamiflu)	**A/B**	**A/B**	**> 1***
zanamivir INH (Relenza)	**A/B**	**A/B**	**> 7***

*Relenza not for children < 7yo but Tamiflu can be used for kids from 2 weeks of age

Influenza

Prevention (85% with annual vaccines)

Influenza A/B vaccine for:

- over >50
- any adult or child with chronic medical problems (esp. cardiac & respiratory)
- nursing home residents
- healthcare workers
- pregnant women
- children 6-59 months
- household contacts of those above
- contraindications to vaccines: allergy to eggs, acute febrile illness, thrombocytopenia

Influenza

Watch For…. **<u>Reye's syndrome</u>:**

- children with A/B and VZV treated with salicylates presents with hepatic and CNS complications

Acute Bronchitis (aka tracheobronchitis)

Etiology:

- >90% are viral (rhino, corona, RSV, etc.)
- inflammation of airways (trachea, bronchi, bronchioles) characterized by cough

Clinical Findings:

- **cough *with or without*** sputum (color not predictive of bacteria), fever, or substernal discomfort
- expiratory rhonchi or wheezes

Labs/Diagnosis:

- CXR: absence of markings
- *but*…..true bronchitis difficult to distinguish from pneumonia…get a CXR!

Acute Bronchitis (*aka tracheobronchitis*)

Treatment:

antibiotics are only indicated for the following:

- elderly
- cardiopulm. diseases plus cough > 7-10 d
- immunocompromised

What is appropriate?

- treat symptoms (OTC meds +/-)
- bronchodilators if airflow obstruction

cough can persist

- 3 weeks in 50% of patients
- >1 month in 25%

Community Acquired Pneumonia (CAP)

Background:

- #1 infectious cause of death in US; #8 overall
- generally acquired via aspiration of previously colonized upper airway
- acquired in the home or non-hospital environment

Etiology:

- bacteria isolated more than viruses (e.g. influenza, RSV, adenovirus, parainfluenza)
- *S. pneumo* (m.c. bacterial) >*H. influenza* > *M. cat*
- Atypicals: *Legionella, Mycoplasma*, & *Chlamydia*

Community Acquired Pneumonia (CAP)

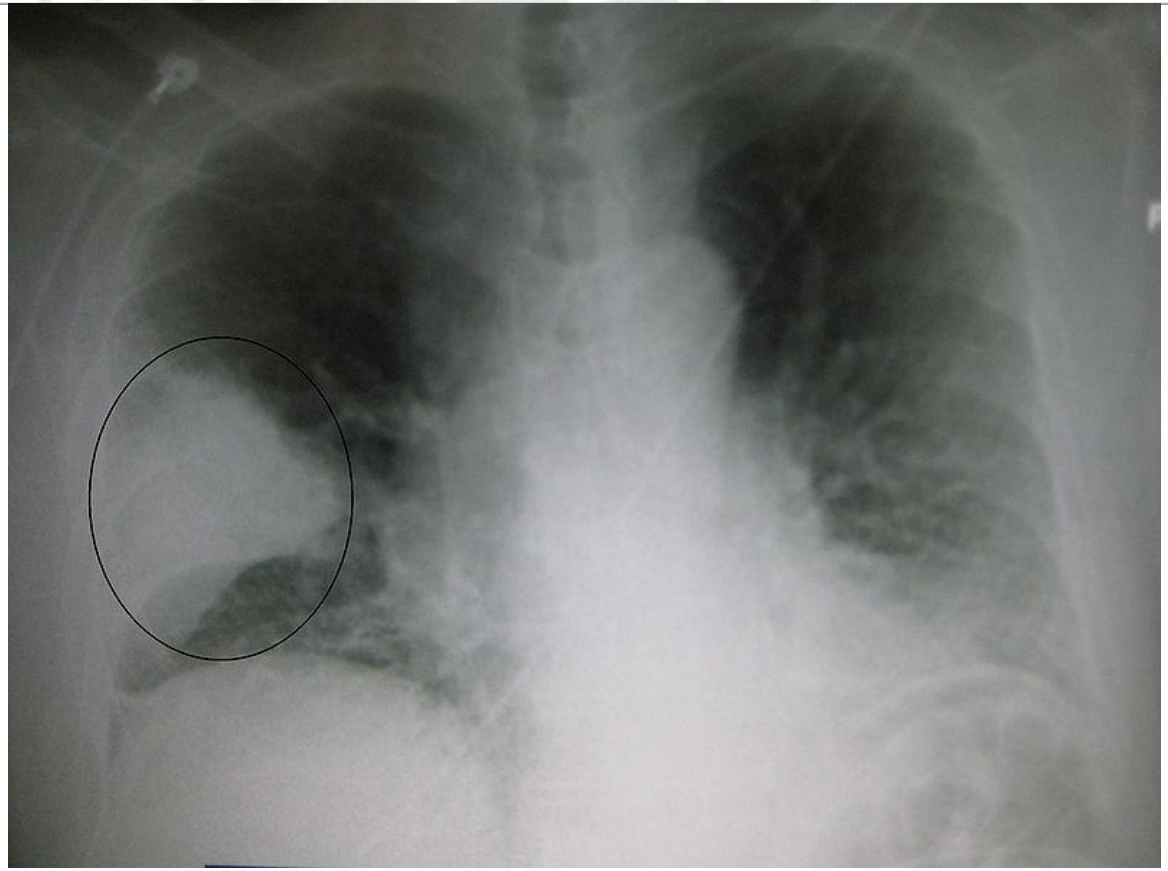
Clinical Findings:

- most common signs are tachycardia/tachypnea
- fever/hypothermia; rigor/sweats;
- new cough + / — sputum, dyspnea
- altered breath sounds/rales
- dullness to percussion with consolidation/effusion
- *but*.....chest exam alone not accurate to confirm/exclude diagnosis

Community Acquired Pneumonia (CAP)

Labs/Diagnosis: ***clinical diagnosis!***

- sputum gram stain
- sputum C&S (less sensitive and specific)
- CXR:
  - patchy, segmental lobar, multi lobar consolidation
  - no pattern is pathognomonic
- Ag studies (*Legionella, Pneumococcus*, respiratory viruses)
- blood cultures x 2 (if hospitalized)
- (?) procalcitonin to distinguish viral vs bacterial



A very prominent pneumonia of the middle lobe of the right lung

Source: http://en.wikipedia.org/wiki/File:PneumonisWedge09.JPG

Community Acquired Pneumonia (CAP)

Treatment:

outpatient:
- doxycycline, erythromycin, macrolides (clarithro >> azithro)
- respiratory fluoroquinolones (if comorbid conditions)

inpatient:
- coverage of *S. pneumo* and *Legionella*
- ceftriaxone (cefotaxime) plus macrolide
- respiratory fluoroquinolones (non ICU) eg levofloxacin, moxifloxacin

hospitalization for CAP?

- clinical judgment
- PORT classification
- consider if: age > 50 with co-morbidities, altered mental status, or hemodynamically unstable

Community Acquired Pneumonia (CAP)

Prevention:
- pneumococcal vaccine
- age > 65 or co-morbid conditions

Remember…

- Expect improvement in 48 -72 hrs. with the right antibiotic
- CXR may worsen but patient improves clinically
- Fever can last 2-5d with pneumococcus; longer with others
- Rales can persist > 7 days in up to 40% of patients
- CXR may not clear for several weeks
- If patient not responding to initial therapy…

  consider: virus, TB, resistant organism, *Pneumocystis* or non- infectious illness

Community Acquired Pneumonia (CAP)

M. pneumoniae -low grade fever -cough -bullous myringitis -cold agglutinins ***P. jiroveci (PCP)*** -slow onset -increased LDH -more hypoxemic than CXR seems -"ground glass" infiltrates **rats** *-Y. pestis*	***L. pneumophila*** -hyponatremia -diarrhea ***C. psittaci*** -psittacine birds -Zoonotic disease ***S. pneumoniae*** -single rigor -rust colored sputum	**alcoholics** *-K. pneumoniae:* currant jelly sputum (dark red mucoid) **cystic fibrosis** *-Pseudomonas* **college student** *-Mycoplasma* *-Chlamydia* **air conditioning / aerosolized water** *-Legionella* **HIV/AIDS** *- P. jiroveci*	**rabbits** *-F. tularensis* **post splenectomy** -encapsulated organism *-S. pneumo* *-H. flu* **leukemia** -fungus **children < 1 year** *-RSV* **children 2-5 years** *-parainfluenza* **COPD** *-H. flu*

Pneumonia: Nosocomial (HCAP)

Background:

- onset of pneumonia > 72 hours after admission
- highest risk: ICU patients on ventilation
- #2 cause of hospital acquired infection
- mortality from 20-50%

Etiology:

- variable geographically
- based on patient risk factors
- Pseudomonas, *S. aureus,* Klebsiella, *E. coli,* *Enterobacter*

Pneumonia: Nosocomial (HCAP)

Clinical Findings (same as CAP):

- most common signs are tachycardia/tachypnea
- fever/hypothermia; rigor/sweats; new cough +/− sputum, dyspnea
- altered breath sounds/rales; dullness to percussion with effusion

Diagnosis (CDC use for epidemiology):

- onset in 72 hours
- PE with rales/dullness or infiltrate on CXR
- one of following:
  - purulent sputum
  - isolated pathogen
  - Ab titers
  - histopathologic evidence of pneumonia

Pneumonia: Nosocomial (HCAP)

Labs/Diagnosis:

- blood culture
- sputum +/-
- CXR evidence of new infiltrate if VAP

Treatment:

- varies with organism, CXR findings, and Abx sensitivities; NO UNIFORM CONSENSUS
- empirical initially with broad coverage
  - cefepime
  - ticarcillin/clavulanic acid
  - piperacillin/tazobactam
  - meropenem

Pneumonia: HIV Related

Background:

- *Pneumocystis jiroveci* (formerly PCP)

Etiology:

- most common opportunistic infection assoc. with AIDS (CD4 < 200)
- also occurs in patients with CA, malnourished, immunosuppressed

Clinical Findings:

- typically sub acute in presentation
- fever, tachypnea, SOB, non-productive cough

Pneumonia: HIV Related

Labs/Diagnosis:

- difficult to diagnose due to non-specific symptoms (fever, cough, SOB)
- CXR: cornerstone of diagnosis with diffuse or peri-hilar infiltrates; *no effusions seen*
- lymphopenia with low CD4 count
- sputum if possible to isolate the organism
- bronchoalveolar lavage

Treatment: TMP/SMX (or pentamidine, atovaquone, others)

Other:

- extremely high mortality (near 100%) if not tx
- primary prophylaxis:

  TMP/SMX

  all AIDS patients with CD4 < 200

Tuberculosis

Background:

- overall, 10% infected with TB will develop the disease
- Primary TB
  - 95 % become Latent TB Infection (LTBI)
    - not considered infectious
    - cannot spread TB
    - asymptomatic
    - *but* have inactive TB in their body
  - 5% become Progressive Primary TB (PPTB)
- Secondary
  - usually reactivation TB develops from LTBI

Etiology: *M. tuberculosis:* transmitted by resp. droplets

Tuberculosis

Clinical Findings:

- may be asymptomatic
- ***cough is most common symptom***
- classic symptom complex: fever, drenching night sweats, anorexia, weight loss
- common pulmonary symptoms

  cough, pleuritic chest pain, SOB, hemoptysis

  post-tussive rales are classic

Diagnosis:

- CXR, sputum culture, acid fast stain of sputum smear

  **organism necessary to obtain susceptibilities*

Tuberculosis

Labs: Sputum: AFB

PPD: measure induration, *not erythema*

positive indicates exposure not necessarily active disease

CXR: Primary:

- homogeneous infiltrates
- hilar/paratracheal lymph node enlargement
- segmental atelectasis
- cavitations with progressive disease (PPTB)

Reactivation:

- fibrocavitary apical disease, nodules, infiltrates
- posterior and apical segments of RUL
- apical-posterior segments of LUL
- superior segments of LL
- miliary pattern in hematogenous dissemination

Tuberculosis

Ghon/Ranke complexes: healed primary infection

Biopsy:

- caseating granulomas (aka necrotizing granulomas) is the histologic hallmark

Miscellaneous:

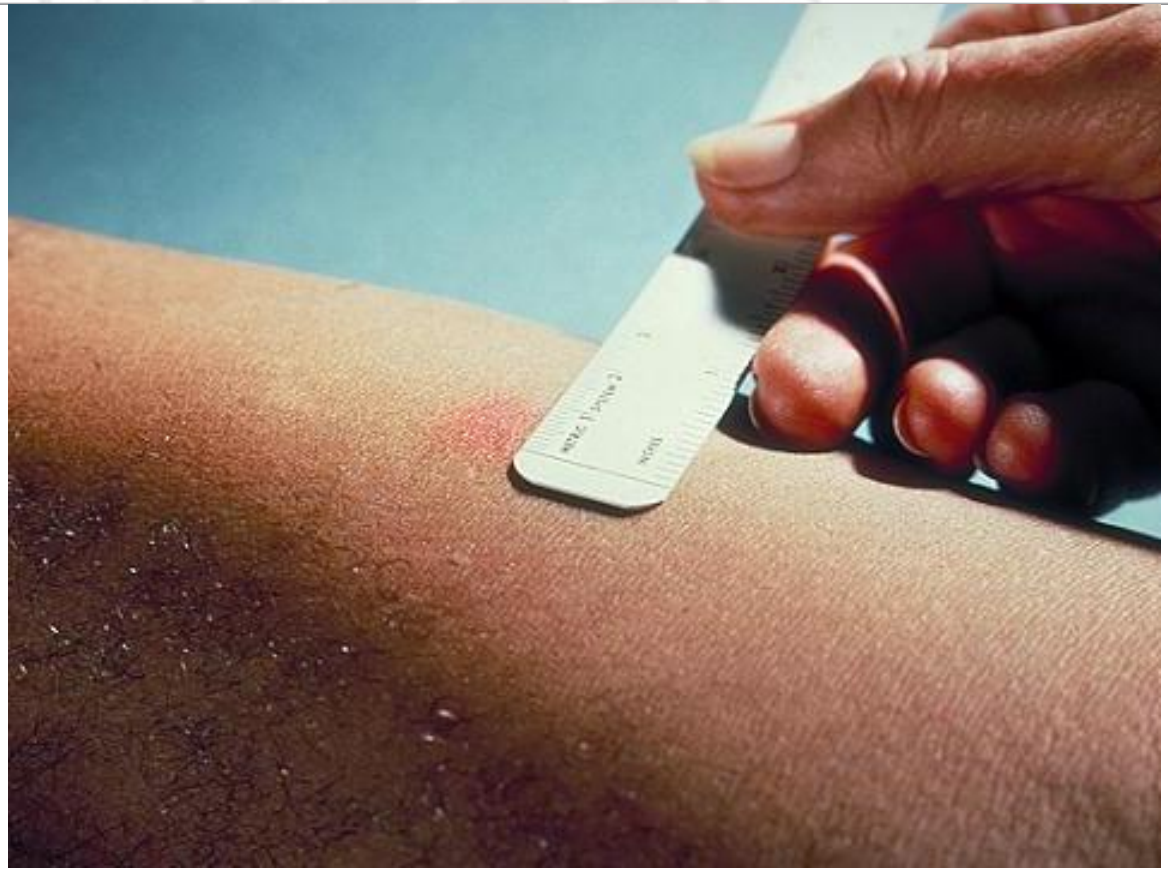
- Pott's Disease:
  - extrapulmonary TB (tuberculous spondylitis)
  - m.c. in thoracic spine

An AP CXR of a patient with advanced bilateral pulmonary tuberculosis. It reveals the presence of bilateral pulmonary infiltrate (white triangles), and "caving formation" (black arrows) present in the right apical region.

Source: http://phil.cdc.gov/phil/home.asp ID#: 2543 US Department of Health and Human Services

Tuberculosis

Classification Of Positive Tuberculin Skin Test Reactions	
Reaction Size	**Group**
≥ 5 mm	1. HIV positive persons 2. Recent contacts of those with active TB 3. Persons with evidence of TB on CXR 4. Immunosuppressed patients on steroids
≥ 10 mm	1. Recent immigrants from countries with high rate of TB infection 2. HIV negative injection drug users 3. Mycobacteriology lab personnel 4. Residents/Employees of high risk congregate settings 5. Persons with certain medical conditions: DM, silicosis, CRF, etc. 6. Children < 4 years of age 7. Infants, children, adolescents exposed to adults at high risk
≥ 15 mm	1. Persons with no risk factors for TB



Measure induration (not erythema) at 48-72 hours

http://en.wikipedia.org/wiki/File:Mantoux_test.jpg

Tuberculosis

Treatment:

- LTBI: (treat only after active TB is ruled out!)
  - *INH x 9 months *or*
  - INH and RPT (DOT) x 3 months *or*
  - *RIF x 4 months (only if in contact with TB resistant persons)
- Active TB:
  - INH/RIF/PZA/EMB x 2 months…then, INH/RIF x 4 months (if sensitive)

Tuberculosis

Anti-tuberculous class specific side effects

INH	→	hepatitis, peripheral neuropathy co-administer Vitamin B6 (pyridoxine)
RIF	→	hepatitis, flu syndrome, orange body fluids
EMB	→	optic neuritis, red-green vision loss

Tuberculosis

Treatment Considerations

- Multiple drugs are necessary
- Sensitivity testing is important
- Single daily dose is effective
- Prolonged treatment may be necessary
- Ensure compliance

Epiglottitis (supraglottitis)

Etiology: viral or bacterial

Signs/Symptoms: rapidly developing sore throat ***or*** odynophagia out of proportion to clinical findings

Labs: laryngoscopy, lateral films (thumb print sign)

Treatment: ceftizoxime or cefuroxime; +/- dexamethasone

Pertussis

Background:
- usually affects infants and young children
- incidence increasing in adults (27% of all cases)
- protection from childhood vaccines wears off

Etiology:
- *Bordatella pertussis*
- transmitted via respiratory droplets
- incubation 6-20 days (most often 7 days)

Clinical Findings:
- resembles common cold/bronchitis
- "whoop" rare in adults

Pertussis

Labs:	• PCR is current diagnostic standard
	• more sensitive then culture
Treatment:	• antibiotics to eradicate organism *but does not alter course of illness*
	• erythromycin, azithromycin, clarithromycin or TMP-SMX
Prevention:	• vaccination with Tdap (instead of Td)
Prophylaxis:	• same as treatment when given within 3 weeks of onset of cough in index case

Question 1 of 2

A 72 year-old male patient presents c/o acute onset of fever, with pleuritic chest pain, a single rigor, and rust colored sputum. CXR is normal. What is the most likely etiologic agent?

- H. influenzae
- K. pneumoniae
- M. pneumoniae
- P. aeruginosa
- S. pneumoniae

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Part II:
Neoplastic Diseases

- Pulmonary Nodules
- Bronchogenic Carcinoma
- Carcinoid Tumors
- Metastatic (Secondary) Tumors

Solitary Pulmonary Nodule

Background:

- a.k.a. coin lesion, lung nodule
- lesion < 3cm (if > 3cm = "mass")
- 40% are malignant

Etiology:

- ***most are infectious granulomas:*** (old or active TB, fungal infection, foreign body reaction)
- carcinoma
- hamartoma
- metastasis (usually multiple)
- bronchial adenoma (95% are carcinoid tumors)

Solitary Pulmonary Nodule

Clinical Findings: most are asymptomatic

Labs/Diagnosis:

CXR: • lesion < 3 cm, isolated, rounded opacity surrounded by normal lung

• old radiographs for comparison?

compare size (doubling time)

larger 30-500 days → malignancy

rapid growth < 30 days → infection

no growth in 2 yrs → benign

Solitary Pulmonary Nodule

Labs/Diagnosis:

CT: • determine nature, location, progression, extent evaluate with CT and biopsy for diagnosis

smooth, well defined	→	often benign
ill defined/lobular	→	suggests CA
spiculated/peripheral halo	→	often CA

Solitary Pulmonary Nodule

Solitary Nodule Comparison		
	malignant	***benign***
age	> 45-50	< 35
calcifications	absent to irregular calcifications	central/uniform/laminated or popcorn like
size	>2cm	<2cm
old films	new or larger	no change
margins	irregular	regular

Solitary Pulmonary Nodule

Treatment:

- >35 years old: resect unless no change in 2 years
- <35 years old, lesion is unchanged, can repeat study in 3-6 months

Bronchogenic CA

Background

- 90% of lung CA is bronchogenic
- leading cause of cancer deaths in men ***and*** women
- *more deaths from lung cancer then colon, breast, and prostate combined!*
- 5-year survival is 15%
- cigarette smoking is #1 risk factor

Bronchogenic CA

Classification Scheme

- SCLC: early mets & aggressive clinical course
  assumes micro metastases at presentation

- NSCLC: (adeno, squamous, large cell)
  slower spreading
  more amenable to treatment (i.e., surgery)

Clinical Findings

- often presents in 50s-70s
- cough, dyspnea, hemoptysis, anorexia, weight loss

Bronchogenic CA

Main histological types

- Squamous (25-35% of cases)
  - keratinization w/ keratin "pearl" formation
  - centrally located, intraluminal mass
  - hemoptysis is common
- **Adenocarcinoma (*m. c. with 35-40% of cases*)**
  - peripheral mass or nodules
- Large Cell (5-10% of cases)
  - heterogeneous group of undifferentiated tumors w/ large cells usually peripherally
  - doesn't fit in other groups
  - fast doubling rates
- Small Cell (15-20% of cases)
  - bronchial origin begins centrally, infiltrating to cause bronchial narrowing/obstruction without a discreet luminal mass

Bronchogenic CA

Labs/Diagnosis:

- cytology & biopsy
- TNM classification only applies to NSCLC

Treatment:

- depends on type/extent of disease
- surgery, chemo, radiation

Other:

- common sites of metastases:
  bone, brain, adrenal glands, liver

Bronchogenic CA

S.P.H.E.R.E. of Lung CA Complications	
SVC Syndrome	compression of SVC: plethora, H/A, mental status changes
Pancoasts Tumor	tumor of the lung apex causes Horner's syndrome and shoulder pain affects brachial plexus & cervical sympathetic n.
Horner's Syndrome	unilateral facial anhidrosis, ptosis, miosis
Endocrine	Carcinoid syndrome: flushing, diarrhea, telangiectasias
Recurrent Laryngeal Symptoms	hoarseness
Effusions	exudative

Bronchogenic CA

Paraneoplastic Syndromes		
Classification	**Syndrome**	**Histological Type**
Endocrine/Metabolic	Cushing's Syndrome SIADH Hypercalcemia Gynecomastia	Small Cell Small Cell Squamous Cell Large Cell
Neuromuscular	Peripheral neuropathy Myesthenia (Eaton-Lambert) Cerebellar Degeneration	Small Cell Small Cell Small Cell
Cardiovascular	Thrombophlebitis	Adenocarcinoma
Hematologic	Anemia DIC Eosinophilia Thrombocytosis	All All All All
Cutaneous	Acanthosis nigricans	All

Carcinoid Tumor

Background:

- aka "carcinoid adenomas", "bronchial gland tumors"
- well-differentiated neuroendocrine tumors
- men = women; usually under age 60
- *m.c. location: GI tract; also lung*

Etiology:

- low-grade malignant neoplasms
- pedunculated / sessile growth in the central bronchi

Carcinoid Tumor

Clinical Findings:
- usually asymptomatic
- localized bronchial obstruction
- hemoptysis, cough, focal wheezing, recurrent pneumonia
- carcinoid syndrome
  - flushing, diarrhea, wheezing, hypotension
  - occurs in 10% of patients

Labs/Diagnosis:
- CT and octreotide scintigraphy for localization
- Bronch / CT → surgery

Treatment:
- surgical excision
- octreotide for symptoms
- most are resistant to radiation and chemotherapy

Mesothelioma

Background:

- primary tumors from pleural lining (80%) or peritoneum (20%)

Etiology: *history of asbestos exposure*

Clinical Findings:

- insidious onset of SOB, non-pleuritic chest pain, weight loss; dullness to percussion, decreased breath sounds, digital clubbing

Mesothelioma

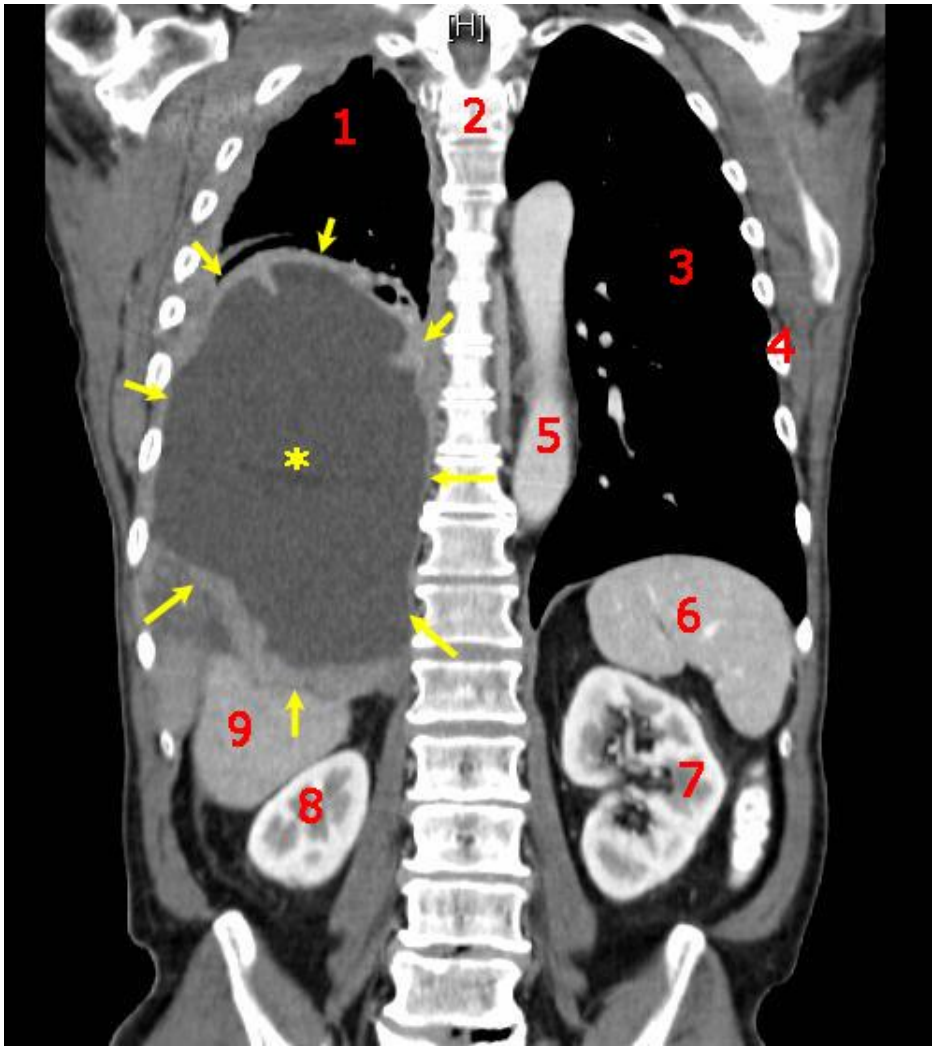
Labs/Diagnosis:

- pleural fluid is exudative and hemorrhagic
- CXR reveals nodular, irregular, unilateral pleural thickening, and effusion
- video assisted thoracic surgery (VATS): biopsy

Treatment:

- none that are effective
- some do chemo/radiation

Other: five year survival is less than 5%



Malignant mesothelioma marked by yellow arrows

Source: http://commons.wikimedia.org/wiki/File:Tumor_Mesothelioma2_legend.jpg

Secondary Lung Cancer

- represents extra-pulmonary metastases
- most frequently primary's that metastasize to lung:
  breast, liver, colon
- almost any CA can spread to lungs
- imaging reveals multiple nodules/masses
- diagnose and treat the primary tumor

Question 1 of 2

Which of the following is the most common type of lung cancer?

- Adenocarcinoma
- Bronchoalveolar
- Large cell
- Small cell
- Squamous cell

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Part III:
Obstructive Pulmonary Disease

- Asthma
- Bronchiectasis
- Chronic Bronchitis
- Emphysema

Obstructive Pulmonary Disease

↓ FEV/FVC

Normal / ↑ TLC

Asthma

Background:

- "reversible" airway condition
- characterized by:
  - acute inflammation
  - bronchial hyper reactivity
  - mucus plugging
  - smooth muscle hypertrophy

Atopy is the strongest identifiable factor:

Atopic "triad": *wheeze*, *eczema*, *seasonal rhinitis*

Asthma

Etiology:

Precipitants: allergens (esp. dust and dust mites), exercise, URI, post nasal drip, GERD, meds (**beta blocker**, ACEI, **ASA**, NSAIDS), stress, cold air

Clinical Findings:

- episodic/chronic symptoms of airway obstruction
- breathlessness, cough, wheeze
- *1/3 of children have no wheeze*
- prolonged expiration/diffuse wheeze

Asthma

Classification Of Severity			
	Symptoms	**Nighttime Symptoms**	**Lung Function**
Intermittent (aka "controlled")	≤ 2x/week	≤2x/month	FEV1 > 80% predicted FEV1/FVC normal
Mild Persistent	>2x/week but not daily	3-4x/month	FEV1 > 80% predicted FEV1/FVC normal
Moderate Persistent	Daily Daily use of beta agonist	>1x/week but not nightly	FEV1 > 60% but < 80% pred. FEV1/FVC reduced 5%
Severe Persistent	Throughout the day	Often 7x/week	FEV1 < 60% pred. FEV1/FVC reduced > 5%

Asthma

Labs/Diagnosis:

- ABGs: mild hypoxia and respiratory alkalosis
- Peak Flow: diminished
- CBC: eosinophilia
- CXR: hyperinflation
- spirometry (pre and post therapy)
  decreased FEV1/FVC (<75%)
- ***definitive test:*** methacholine challenge test
  (aka bronchial provocation test)
  FEV1 decreases by > 20%
- ≥ 10% ↑ FEV with bronchodilator therapy

Asthma

Treatment:

- General
  - remove irritants
  - education on peak flow measurements
  - desensitization
  - oxygen
- Pharmacological
  - Quick relief meds
    - INH beta 2 agonists (e.g. albuterol)
    - glucocorticoids (e.g. prednisone)
    - anticholinergics (e.g. ipratropium)

Asthma

Long-term control therapy

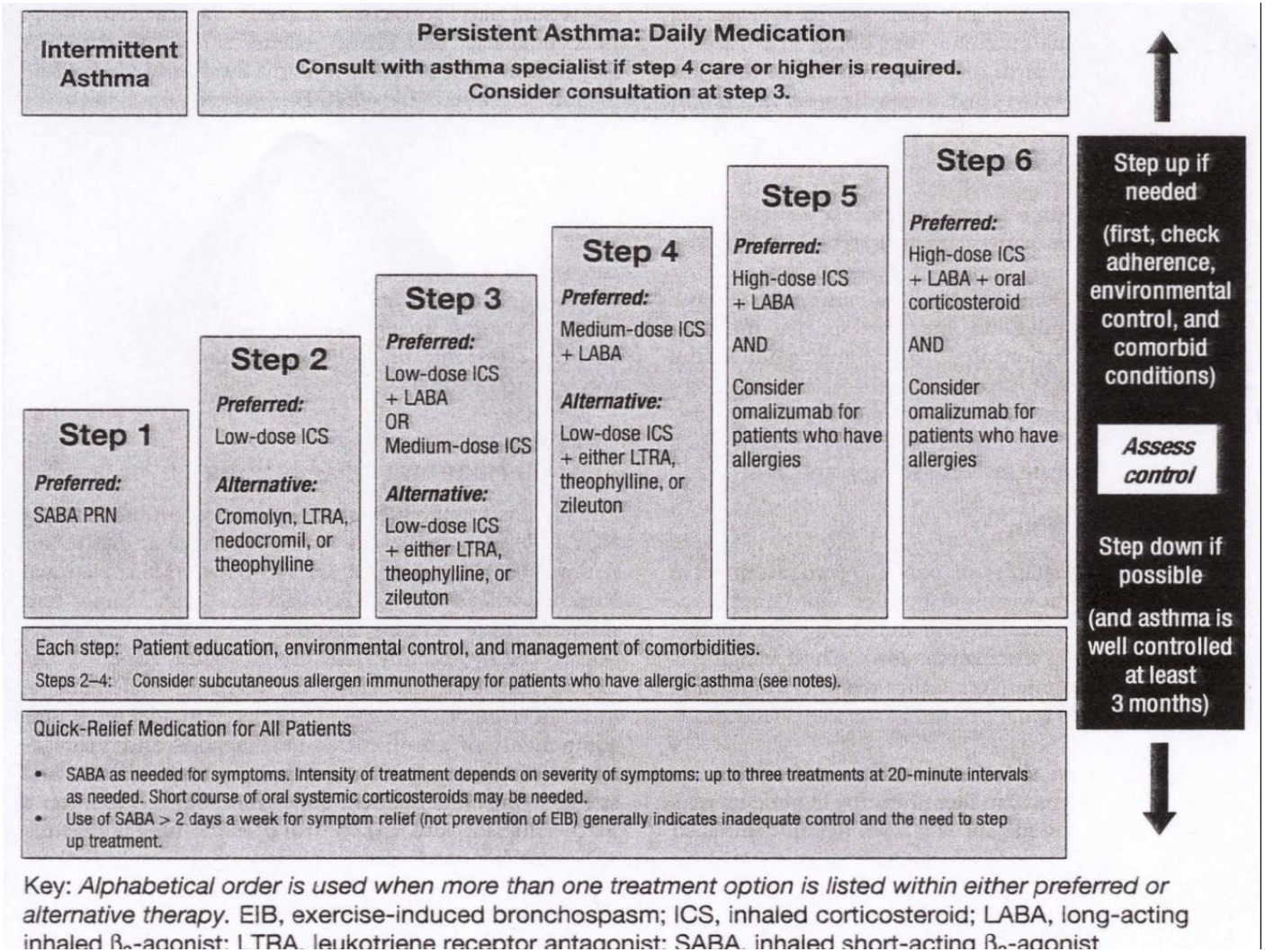
- INH steroids (e.g. fluticasone, budesonide)
  mainstay for **persistent** asthma
- Long acting bronchodilators (LABA):
  (e.g. salmeterol, formoterol)

  INH mast cell stabilizers (e.g. cromolyn)

  INH beta 2 agonists (e.g. salmeterol):

  good for mild persistent or EI asthma

  leukotriene inhibitors (e.g. montelukast (Singulair))
- phosphodiesterase inhibitors (e.g. theophylline)



National Asthma Education and Prevention Program, Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma, NIH Pub No 08-4051, 2007

Bronchiectasis

Background:

- permanent dilation/destruction of the bronchial walls

Etiology:

- congenital: Cystic Fibrosis
- acquired: recurrent infections (TB, fungal infection, lung abscess) obstruction (tumor)

Clinical Findings:

- foul breath, chronic cough with purulent sputum, hemoptysis, recurrent pneumonia, weight loss, anemia, persistent basilar crackles

Bronchiectasis

Labs/Diagnosis:

- Sputum smear/culture
- ***CXR: tram track lung markings***
  honeycombing
  atelectasis
- CT (HRCT): diagnostic test of choice
  thickened bronchial walls with dilated airways

Clinical diagnosis with radiological support

Bronchiectasis

Treatment:

- ambulatory oxygen
- aggressive antibiotics (10-14 days):
  - guided by sputum cultures *or*
  - empiric therapy
    - amoxicillin
    - amox-clavulante (Augmentin)
    - TMP/SMX (Bactrim)
    - ciprofloxacin
- INH bronchodilators for maintenance and acute exacerbations
- lung transplantation

Lung CT with thin slices (1 mm) showing bronchiectasis in the lower lung lobes of a subject with type ZZ alpha-1-antitrypsin deficiency. There are no signs of emphysema.

Source: Fregonese L, Stolk J. Hereditary alpha-1-antitrypsin deficiency and its clinical consequences. Orphanet J Rare Dis. 3, 1, 16. 2008. doi:10.1186/1750-1172-3-16. PMID 18565211.

Source: http://www.flickr.com/photos/pulmonary_pathology/3791074491/ |Date= |Author=Yale Rosen |Permission=[http://creativecommons.org/licenses/by-sa/2.0/ CC-BY-SA 2.0] |other versions= }} [[category:gross patholo)

COPD: Chronic Bronchitis/Emphysema

Background:

- airflow obstruction due to chronic bronchitis or emphysema
- most patients have features of both

Emphysema:

- permanent air space enlargement distal to terminal bronchiole with alveolar wall destruction

Chronic Bronchitis:

- increased bronchial secretions
- cough for > 3 months over at least 2 years

COPD: Chronic Bronchitis/Emphysema

Etiology

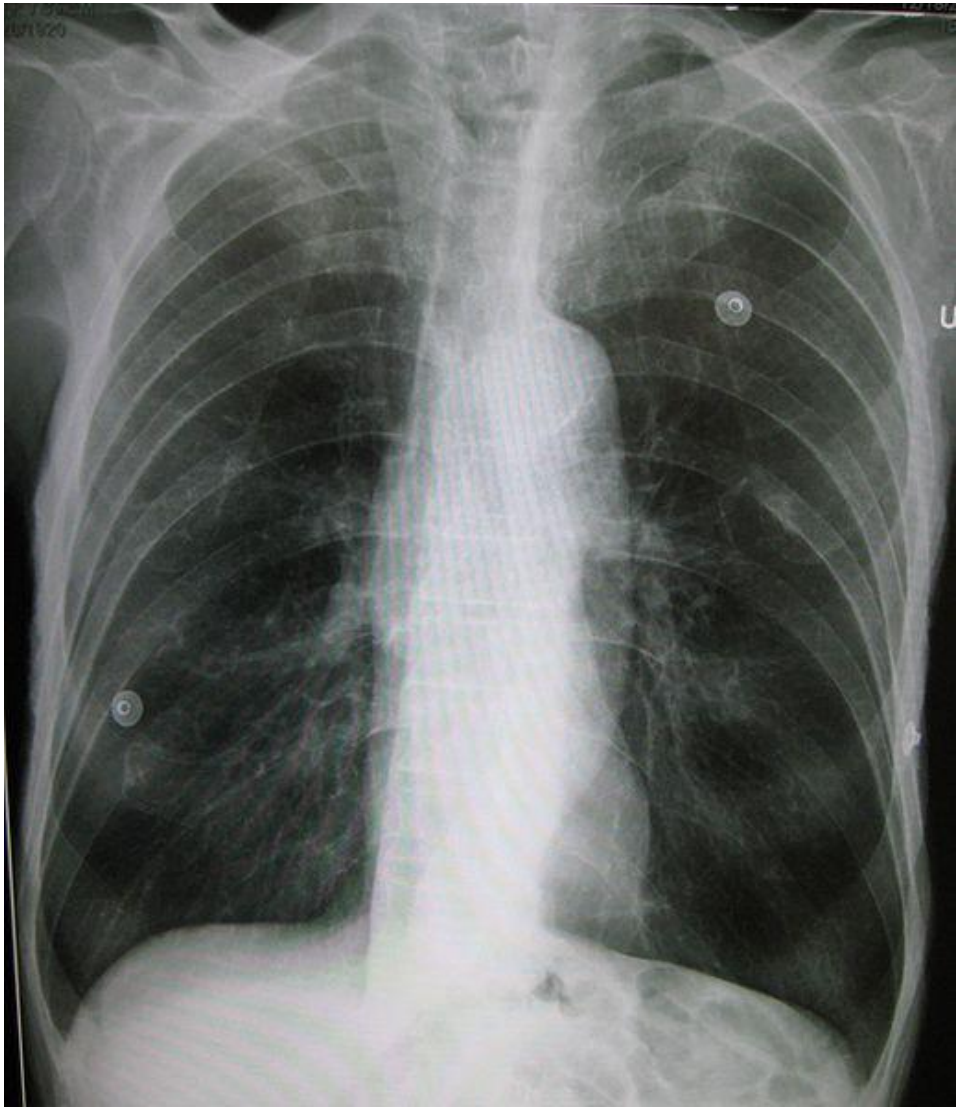
- ***smoking/exposure to tobacco (80%)***
- environmental pollutants
- recurrent URI's
- eosinophilia
- bronchial hyper responsiveness

Labs/Diagnosis:

- PFT: normal early in the disease
  decreased FEV1/FVC occur later
  increased RV and TLC
  confirmed by biopsy
  ↑ Reid index: gland layer is > 50% of total bronchial wall

COPD: Chronic Bronchitis/Emphysema

COPD Comparisons		
	Emphysema Predominant	**Bronchitis Predominant**
Patient type	"pink puffers"	"blue bloaters"
Clinical Findings	***Hallmark: exertional dyspnea*** cough is rare quiet lungs no peripheral edema thin; recent weight loss barrel chest pursed lips breathing hyperventilation	mild dyspnea ***chronic productive cough*** noisy lungs: rhonchi and wheeze peripheral edema overweight and cyanotic
CXR	decreased lung markings at apices flattened diaphragms hyperinflation ***parenchymal bullae and blebs*** small, thin appearing heart	increased interstitial markings at bases diaphragms not flattened



CXR of patient with severe emphysema

Source: http://commons.wikimedia.org/wiki/File:Emphysema2008.jpg

COPD: Chronic Bronchitis/Emphysema

Treatment:

- ***smoking cessation***
- ***oxygen*** *improves the natural history of the disease*
- bronchodilators
  - ***#1: ipatropium***
  - #2: short acting beta agonists: albuterol
  - #3: theophylline
- INH steroids?
- antibiotic: for AECB and acute bronchitis
  - TMP/SMX; augmentin/clavulanate, doxycycline, and others
- influenza and pneumococcal vaccines
- Surgery: transplant, LVRS, bullectomy

Question 1 of 4

Which of the following is the most common cause of chronic bronchitis?

- Air pollution
- Allergies
- Alpha 1 antitrypsin deficiency
- Pneumonia
- Smoking

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Part IV:
Restrictive Pulmonary Diseases

- Idiopathic Pulmonary Fibrosis
- Pneumoconioses
- Sarcoidosis

Restrictive Pulmonary Diseases

- Normal / ↑ FEV/FVC
- ↓ TLC

Idiopathic Pulmonary Fibrosis

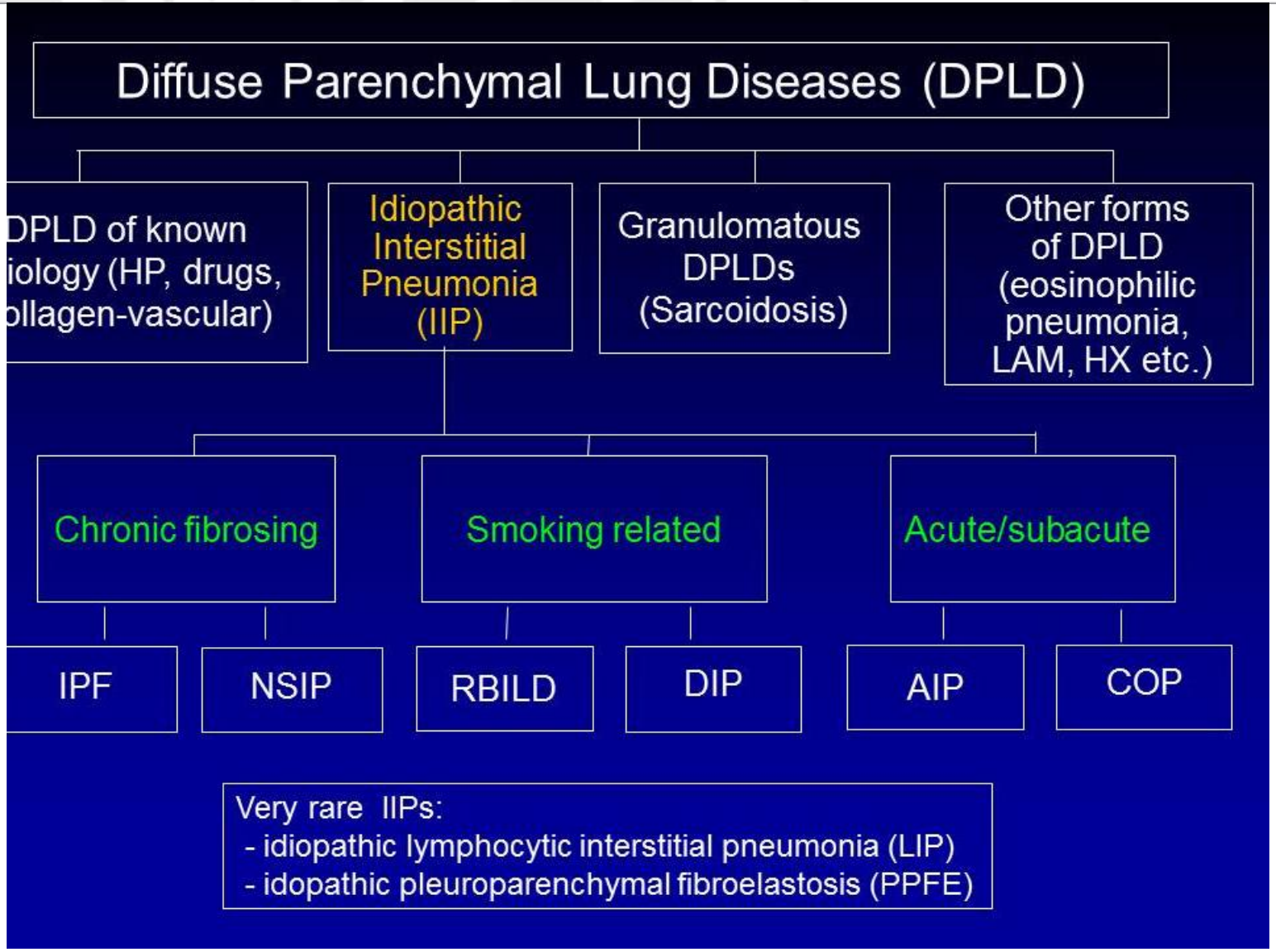
Background:

- most common dx among pts with interstitial lung disease (ILD)
- includes group of distinct histopathologic features
- ensure its truly idiopathic as most ILD are due to infection, drugs, environmental/occupational exposures

Etiology: unknown

Clinical Findings:

- insidious dry cough
- exertional dyspnea
- diffuse, fine, end insp. crackles/rales ("velcro" at bases)
- clubbing



American Thoracic Society/European Respiratory Society International Multidisciplinary Consensus Classification of the Idiopathic Interstitial Pneumonias. (2013). Am Respir Crit Care Med. 188 (6): 733–748. PMID 24032382.

Idiopathic Pulmonary Fibrosis

Labs/Diagnosis:

CXR/HRCT:

- low lung volumes
- patchy, diffuse fibrosis
- pleural honeycombing
- biopsy helps to exclude other causes

Treatment:

- *controversial*
- corticosteroids
- interferon

Pneumoconioses

Chronic lung diseases

Differentiated by origin of the precipitating agent

Etiology:

- generally industrial
- inhalation of mineral or metal dusts
- fibrotic lung develops progressively from ingestion of the agents by macrophages leading to cell injury and death

Treatment:

- generally supportive

Pneumoconioses

Comparison of Pneumoconioses			
Disease	**Occupation**	**Diagnosis**	**Complications**
asbestosis	insulation, demolition, construction,	BX: asbestos bodies CXR: linear opacities at bases and pleural plaques	increased risk of lung CA and ***mesothelioma***, esp. if smoker
CWP	coal miner	CXR: nodular opacities at upper lung fields	progressive massive fibrosis
silicosis	miners, sand blasters, quarry workers, stone workers,	CXR: nodular opacities at upper lung fields	increased risk of ***TB***; progressive massive fibrosis
berryliosis	high technology fields: aerospace, nuclear power, ceramics, foundries, tool & die manufacturing	CXR: diffuse infiltrates and hilar adenopathy	needs chronic steroids

Sarcoidosis

Background:

- ↑ incidence in North American blacks & Northern European whites

Etiology:

- systemic disease of unknown etiology

Clinical Findings:

- malaise, fever, slowly progressing dyspnea, cough
- pulmonary findings are limited
- extra-pulmonary findings common:
  - erythema nodosum
  - parotid gland enlargement

Sarcoidosis

Labs:
- ACE levels elevated
- CXR → bilateral hilar adenopathy
- hypercalcemia

Diagnosis: biopsy shows <u>non-caseating granulomas</u>

Treatment: prednisone

Other: "**GRUELING**"

Granulomas
RA
Uveitis
Erythema nodosum
Lymphadenopathy
Interstitial fibrosis
Negative TB test
Gammaglobulinemia

Question 1 of 2

Which of the following is an example of a restrictive lung disease?

- Asthma
- Bronchiectasis
- COPD
- Cystic fibrosis
- Sarcoidosis

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Part V:
Pleural Diseases

- Pleural Effusion
- Pneumothorax

Pleural Effusions

Background

- abnormal fluid collection in the pleural space
- 25% of effusions are associated with malignancy

Important to distinguish transudate from exudate

Video Placeholder
Your video will display here.

Pleural Effusions

Etiology: 5 types of effusions

- exudates: ***"leaky capillaries"***
  - these three cause 80%
    - para-pneumonic
    - malignancy
    - PE (but 20% as transudate)
  - infection (TB), malignancy, trauma
- transudates: ***"intact capillaries"***
  - CHF (90%), atelectasis, renal/liver ds. (cirrhosis)
- empyemas: direct infection of an exudate
- hemothorax: trauma
- chylothorax: TB

Pleural Effusions

Clinical Findings:

- often asymptomatic

  progressive dyspnea on exertion and pleuritic chest pain

- presentation is variable

  asymptomatic → small effusion

  dyspnea/cough → large effusion

- *percussion dullness*
- decreased tactile fremitus
- diminished/absent breath sounds
- b/l (transudates) vs. unilateral (exudates)

Pleural Effusions

Labs:

imaging helps define extent of effusion

- lateral decubitus (free flowing vs. loculated fluid)
- upright (blunting of costophrenic sulcus)
- CT scan for small effusions

Diagnosis:

thoracentesis is the gold standard

- send for protein, LDH, pH, total & cell counts, glucose
  cytology?
- Gram stain with C & S?

Massive Left-Sided Pleural Effusion

Source: http://en.wikipedia.org/wiki/Image:Left-sided_Pleural_Effusion.jpg

Pleural Effusions

Treatment:

Transudates:

- correct underlying condition
- therapeutic thoracentesis if severe dyspnea

Exudates:

- drainage for empyemas
- pleurodesis for malignant pleural effusions

Pleural Effusions

Other:

transudates vs. exudates **(Light's criteria)**

exudate if meets any *one* of the following:

- pleural fluid protein/serum protein ratio > 0.5
- pleural fluid LDH / serum LDH ratio > 0.6
- pleural fluid LDH > 2/3 upper limit of normal for serum LDH (a cut-off value of 200 IU/L was used previously)

Pneumothorax

Background: accumulation of air in pleural space

Etiology/Classifications:

- Spontaneous (1° or 2°)
  - Primary: (PSP) occurs in absence of underlying ds.
    - tall, thin males (rupture of apical blebs)
  - Secondary: (SSP) underlying ds.
    - *COPD*, asthma, CF, ILD
- Traumatic: penetrating/blunt trauma (incl. iatrogenic)
- Tension pneumothorax: ***medical emergency!***
  - penetrating trauma, CPR, pos pressure ventilation:
    - lung collapse
    - → contra lateral mediastinal shift
    - → hypotension 2° impaired v. return

Pneumothorax

Clinical Findings:

Spontaneous:

- ipsilateral, unilateral chest pain, sudden and pleuritic, dyspnea, cough
- absent/diminished breath sounds
- hyper resonance
- decreased tactile fremitus
- if small, exam is unimpressive

Tension: (in addition to above…)

- respiratory distress, falling SaO2,
- hypotension, distended neck veins, tracheal deviation

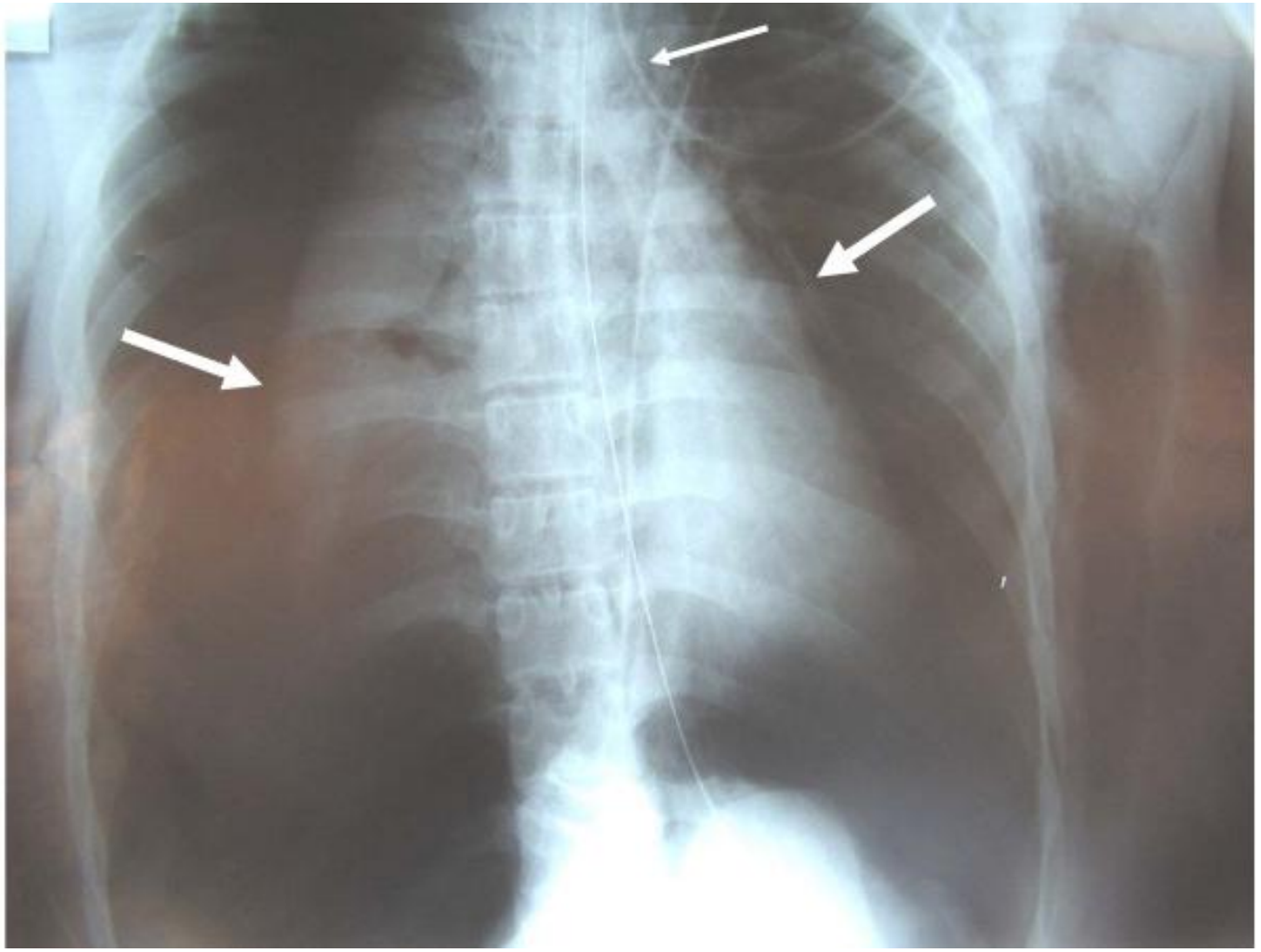
Pneumothorax

Labs/Diagnosis:

- ***end expiratory chest film reveals visceral pleural air***
- Tension: air on affected side with contralateral mediastinal shift

Treatment:

- Primary spontaneous/secondary:
  - <15% diameter of hemithorax on CXR: rest, cough, chest pain relief, serial CXRs
  - >15%: chest tube plus above measures
- Tension: immediate needle decompression 2nd ICS at MCL



Bilateral pneumothorax (larger arrows)

Source: Le Guen et al. Critical Care 2007 11:R94 doi:10.1186/cc6109 http://ccforum.com/content/11/5/R94

Left tension pneumothorax

Source: http://clinicalcases.blogspot.com/2004/02/tension-pneumothorax.html

Question 1 of 2

A 54 year-old patient presents with acute onset of chest pain, SOB and hypotension. CXR reveals a mediastinal shift to the right. Which of the following is the most appropriate next step?

- Admit to hospital for observation
- Insert an airway
- Order a pulmonary consult
- Perform immediate needle decompression

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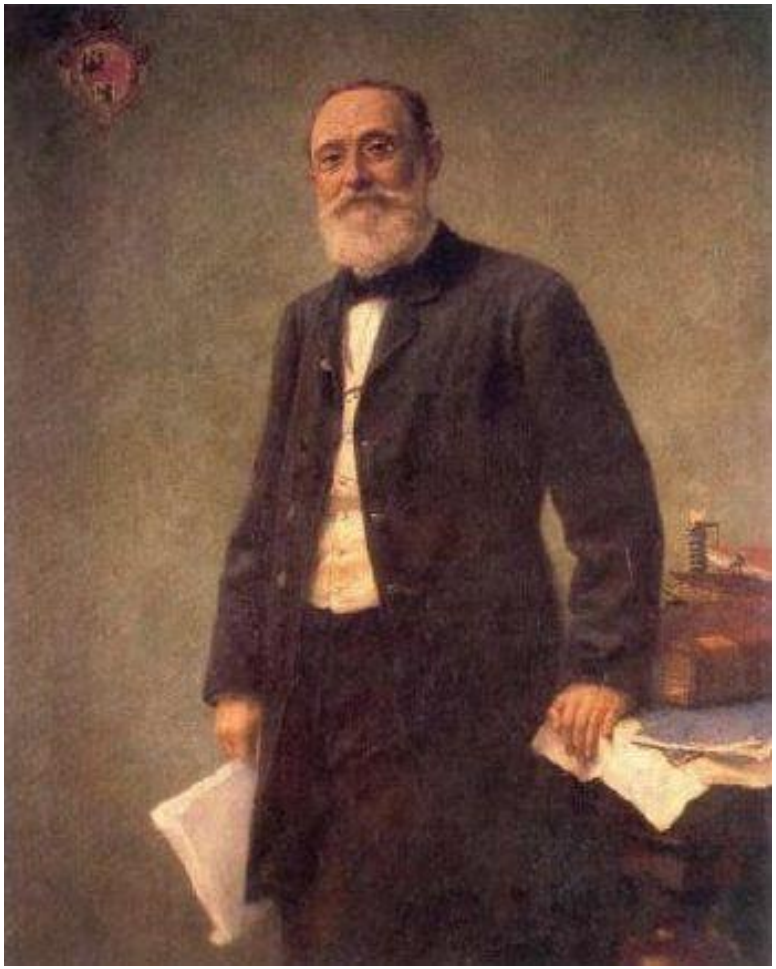
Part VI: Pulmonary Circulation

- Pulmonary Thromboembolism
- Pulmonary Hypertension
- Cor Pulmonale
- ARDS



Rudolf Virchow by Hugo Vogel, 1861

Source: http://www.kunsttexte.de/download/bwt/werner.pdf

Pulmonary Embolism (PE)

Background

- occlusion of pulmonary arterial circulation from an embolized substance
- #3 leading cause of death in hospitalized pts.

Risk Factors: (Dr. Rudolf Virchow's triad)

- hypercoagulable state: (e.g. CA)
- venous stasis (e.g. prolonged rest/cast)
- vascular intimal inflammation / injury (e.g. surgery/trauma)
- surgical procedures: orthopedic, pelvic, abdominal CA, OCPs, pregnancy

Pulmonary Embolism (PE)

Etiology: most are from thrombus

- 95% deep calf veins that propagate proximal to popliteal / ileofemoral veins
- risk of PE greater with proximal thrombus
- others:?

air	→	central lines
amniotic fluid	→	active labor
fat	→	long bone (femur) fx

- negative workup in 25-50% patients for VTE

Pulmonary Embolism (PE)

Clinical Findings:

- Homans' sign: low sensitivity/specificity
  calf pain with passive, forcible, dorsiflexion of foot with knee flexed
- variable, signs and symptoms are non-specific but <3% chance of PE in absence of dyspnea with tachypnea or pleuritic chest pain
- most emboli are clinically silent
- *most common symptom: dyspnea (sudden; 85% with RR>16) / pain on inspiration*
- (*consider PE in any hospitalized pt. w/ acute SOB)
- *most common sign: tachycardia (60% with P > 100)*

Pulmonary Embolism (PE)

Labs/Diagnostic Evaluation:

- ECG: *not diagnostic!*
  - sinus tachycardia (most common)
  - atrial dysrhythmias, PEA
  - S1-Q3-T3 (inverted T wave) is rare (20%)
- ABGs: hypoxemia (but 15% have PaO2 > 80)
- Elevated D-Dimer
  - Plasma levels of degraded fibrinogen
  - ***negative D-Dimer (with low clinical suspicion) → strong evidence against DVT***

Pulmonary Embolism (PE)

CXR (abnormalities may be subtle / absent)

- *m.c. abnormality is atelectasis at bases*
- **Westermark's Sign**: focal oligemia (vasoconstriction) in the embolized zone
- **Hampton's Hump:** (**classic finding**)
  wedge shaped infarct

VQ scans:

- "normal" practically rules out PE
- "abnormal" is non specific
- categorized along with CXR
  normal/very low
  low
  intermediate
  high probability

Chest Spiral CT (with and without contrast agent) showing multiple filling defects of principal branches, due to acute and chronic pulmonary embolism

Source: Cardiovascular Ultrasound 2007, 5:26. doi:10.1186/1476-7120-5-26

Pulmonary Embolism (PE)

- Spiral CT (helical) angiography
  - more sensitive then VQ, but less than pulmonary arteriography
  - less sensitive in the distal segmental arteries
- Pulmonary Arteriography
  - shows intraluminal filling defect
  - **"Gold Standard"**
- LE Venous Doppler (not good for dx of PE)
  - most commonly used
  - incompressible veins (absence of "wink")
  - 90-94% sensitivity in proximal (less in distal)
- Venography
  - **"Gold Standard"** for diagnosis of LE DVT

Pulmonary Embolism (PE)

Treatment:

- anticoagulation: generally 3-6 months
  - heparin (UFH: sig is TID) → coumadin (INR 2-3x normal)
  - LMWH (sig is QD)
- thrombolytic therapy
  - streptokinase, alteplase, urokinase:
    - reserved for hemodynamically unstable patient
    - not generally recommended
- IVC filter
- surgery: only for saddle emboli

Pulmonary Embolism (PE)

Other:

Prevention:

- combination of mechanical & pharm. measures
- early ambulation
- intermittent pneumatic compression
- unfractionated heparin
- warfarin
- ✔LMWH- enoxaparin (Lovenox, Dalteparin)
- target specific anticoagulants?

  rivaroxaban (Xarelto)

Pulmonary Hypertension

Background:

- pulmonary artery pressure rises to a level inappropriate for a given cardiac output
- self-perpetuating once initiated
- women > men
- 30-50 yo

Pulmonary Hypertension

Etiology:

- primary (idiopathic) hypertension (PPH) is rare
- ***most frequently: secondary pulmonary HTN***
  - (COPD, connective tissue disorder esp. scleroderma)
  - increased pulmonary venous pressure
  - constrictive pericarditis, LV failure, mitral stenosis, mediastinal disease compressing pulmonary veins
  - decreased area of pulmonary arterial bed
  - vasoconstriction
  - loss of vessels
  - lung resection, emphysema, ILD, CVD
  - vessel obstruction

Pulmonary Hypertension

Clinical Findings:

- dull/retrosternal chest pain (angina-like), dyspnea, fatigue, effort syncope
- difficult to diagnose early
- signs/symptoms are often related to underlying cause

Labs:

- polycythemia
- EKG: right axis deviation, RVH, RAE, right ventricular strain

Pulmonary Hypertension

Diagnosis: Multifactorial

Work-up:

- CXR/CT: increased vasculature
- PFTs: underlying airflow obstruction or restricted lung volumes
- ECHO: RVH, estimated pulmonary artery pressure
- catheterization to determine degree of HTN
- others (VQ scan, serology: conn tissue disorders, etc.)

Pulmonary Hypertension

Treatment:

underlying cause

- oxygen if from COPD
- anticoagulants if from emboli
- diuretics/salt restriction for cor pulmonale
- vasodilators?

epoprostenol (PGI2)

prostacyclin

Cor Pulmonale

Background:

- Failure of the right side of the heart caused by prolonged high blood pressure in the pulmonary artery (pulmonary HTN) and right ventricle of the heart.
- RV enlargement leads to RV failure

Etiology:

- *if acute, think P.E.; if chronic think COPD*
- pulmonary vascular disease (PE, vasculitis, ARDS)
- respiratory disease
  - obstructive (asthma, COPD)
  - restrictive (ILD, lung resection)

Cor Pulmonale

Clinical Findings:

- fatigue, exertional dyspnea, and syncope with exertion
- increase in chest diameter
- labored resp. efforts with retractions of the chest wall
- hyper resonance to percussion

  diminished breath sounds

  wheezing

  distant heart sounds

  cyanosis (rarely)

Cor Pulmonale

Labs/Diagnosis:

- CXR
- EKG: RAD > 30° ; flat, inverted T waves in RV precordial leads

Treatment:

- oxygen
- decrease pulm. Vasc. resistance and pulmonary HTN
- treat underlying disorder

Acute Respiratory Distress Syndrome

Clinical Definition:

- acute (12-18hours) hypoxemic respiratory failure after a systemic or pulmonary insult **without** heart failure

Physiological Definition

- bilateral diffuse pulmonary infiltrates
- normal PCWP (≤18 mmHg)
- PaO2/FiO2 < 200

Etiology:

- ***most common*** (one-third of patients): ***sepsis***
- others: toxic inhalation, near drowning, aspiration, etc.

Acute Respiratory Distress Syndrome

Clinical Findings:

respiratory distress, tachypnea, fever, crackles, rhonchi

Labs:

CXR:
- diffuse pulmonary infiltrates *that spares the costophrenic angles*
- air bronchograms in 80%
- *normal* heart size

Diagnosis:
- no biochemical tests to define ARDS
- clinical dx that excludes cardiogenic pulmonary edema

Acute Respiratory Distress Syndrome

Treatment:

- underlying cause *plus* supportive care
- support cardiac output with inotropes, cautious fluids
- mechanical ventilation

  PEEP: lowest levels to recruit atelectic alveoli
  - PaO2 > 60
  - FIO2: < 60%
  - SaO2 ≥ 90%

Other:

- ARDS mortality: 30-40%
- ARDS *plus* sepsis mortality: 90%

Question 1 of 2

A 52 year-old patient presents to the ER with pleuritic chest pain, cough, dyspnea, and hemoptysis. On exam she is anxious with tachycardia and tachypnea. Lab work demonstrates and elevated D-dimer. CXR is normal. What is the most likely diagnosis?

- Emphysema
- Myocardial infarction
- Pneumonia
- Pulmonary embolism
- tension pneumothorax

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Good luck!