

Pulmonology

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Certification and Recertification Exam Review
June 6-9, 2011

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 1-4d

Influenza

Clinical Findings:

epithelial necrosis leading to bacterial superinfection (esp. with pneumococcus or *S. aureus*)

abrupt onset, fever, chills, headache, coryza, myalgias (esp. back and legs), sore throat, proteinuria, leukopenia, cervical lymphadenopathy

Diagnosis:

usually clinical (aka presumptive)

rapid Ag tests (nasal/pharyngeal)

fever and cough in areas of epidemic:

positive predictive value of 80%

Influenza

	Prophylaxis	Treatment (w/i 48 hrs)	Ages
amantadine	A	not recommended	≥ 1
rimantadine	A	not recommended	≥ 1
oseltamivir (Tamiflu)	A/B	A/B	$> 1^*$
zanamivir (Relenza)	A/B	A/B	$> 7^*$

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.... Reye's syndrome:

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 + 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; #6 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 effusion

*but.....*chest exam alone not accurate to confirm/exclude
diagnosis

Community Acquired Pneumonia (CAP)

Labs/Diagnosis: *clinical diagnosis!*

sputum gram stain

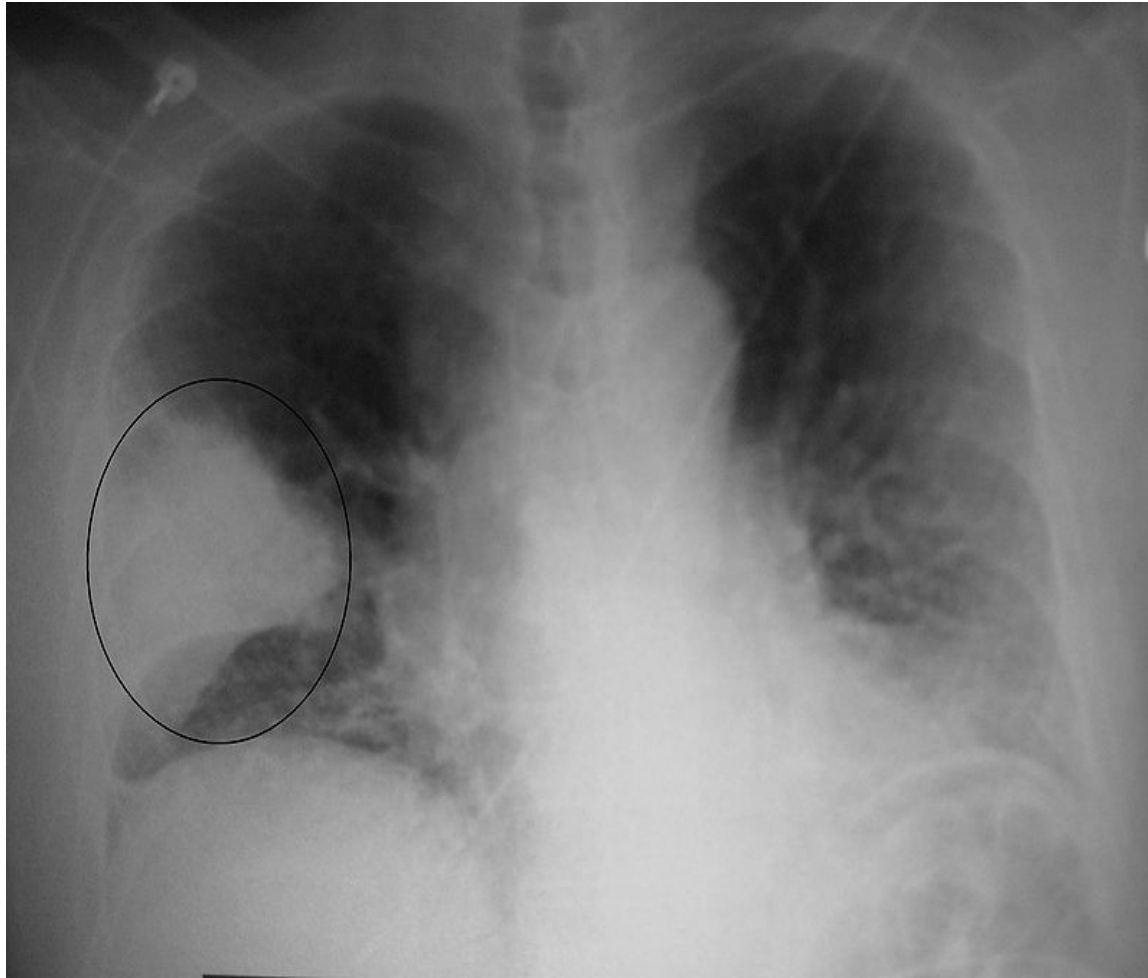
sputum C&S (less sensitive and specific)

CXR: patchy, segmental lobar, multi lobar
consolidation

no pattern is pathognomonic

Ag studies (*Legionella*, *Pneumococcus*,
respiratory viruses)

blood cultures x 2 (if hospitalized)



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
- inpatient: coverage of *S. pneumo* and
Legionella
ceftriaxone (cefotaxime) plus
macrolide
respiratory fluoroquinolones

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)

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

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 ds

CXR: Primary:

homogeneous infiltrates

hilar/paratracheal lymph node enlargement

segmental atelectasis

cavitations with progressive disease (PPTB)

Reactivation:

fibrocavitary apical ds., nodules, infiltrates

posterior and apical segments of RUL

apical-posterior segments of LUL

superior segments of LL

miliary pattern in hematogenous dissemination

Tuberculosis

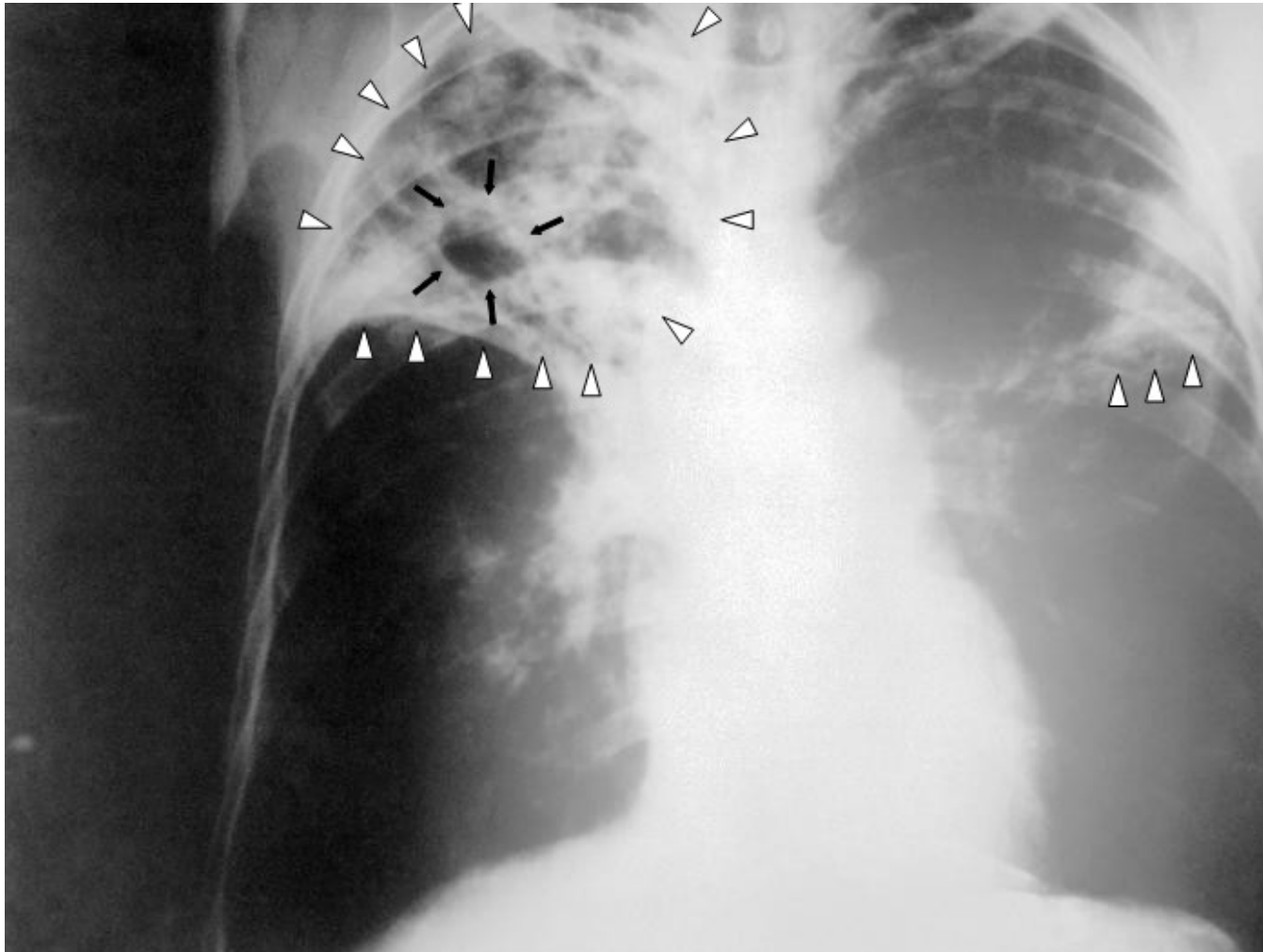
Ghon/Ranke complexes: healed primary infection

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	<ol style="list-style-type: none"> 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	<ol style="list-style-type: none"> 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	<ol style="list-style-type: none"> 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

UMDNJ PANCE/PANRE Review Course

Tuberculosis

Treatment:

LTBI: (treat only after active TB is ruled out!)

INH x 9 months *or*

PZA and RIF x 2 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 is 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)

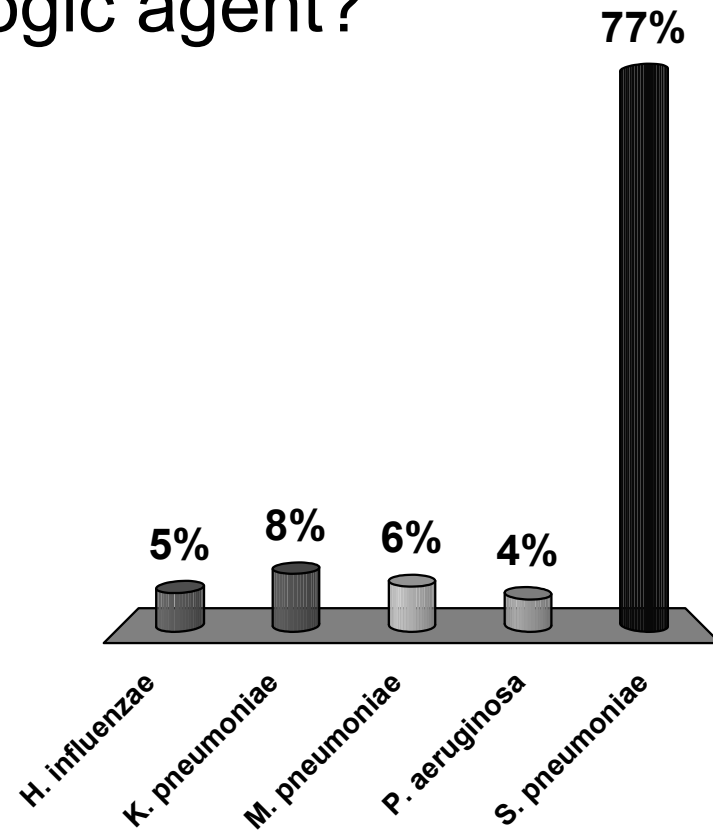
- Clinical Findings:
- resembles common cold/bronchitis
 - “whoop” rare in adults

Pertussis

- Labs:
- PCR is current diagnostic standard
 - more sensitive than 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

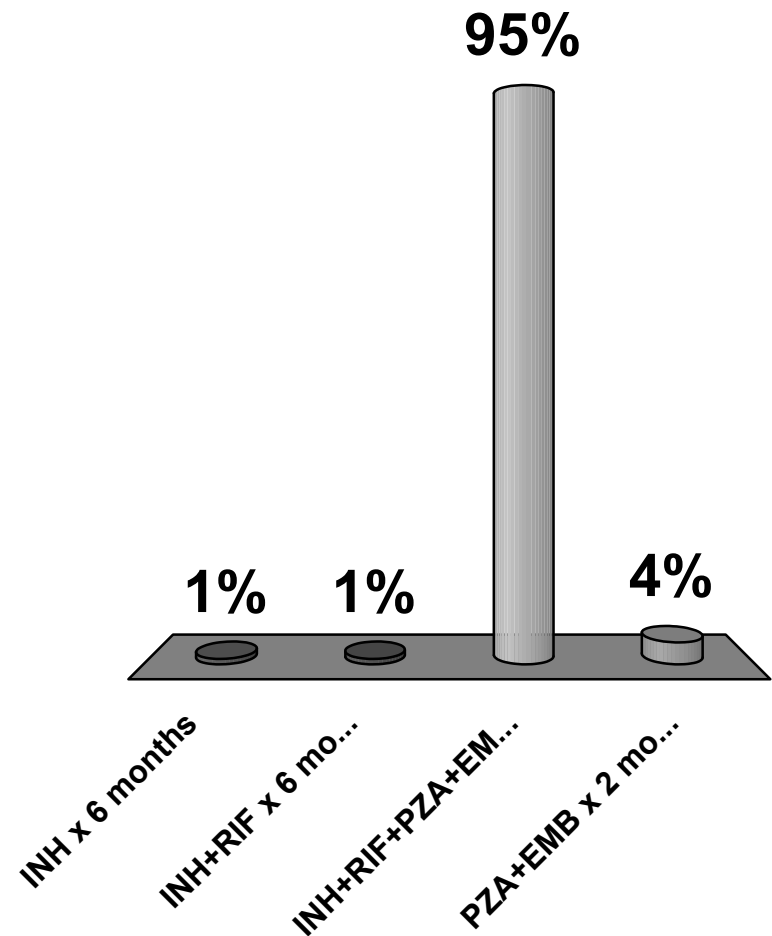
A 72 year-old male patient presents c/o acute onset of fever, with pleuritic chest pain, a single rigor, and rust colored sputum. CXR is normal. What is the most likely etiologic agent?

- A. *H. influenzae*
- B. *K. pneumoniae*
- C. *M. pneumoniae*
- D. *P. aeruginosa*
- E. *S. pneumoniae*



Which of the following regimens is most appropriate for the treatment of active TB in immunocompetent patients?

- A. INH x 6 months
- B. INH+RIF x 6 months
- C. INH+RIF+PZA+EMB x 2 months then INH+RIF x 4 months
- D. PZA+EMB x 2 months then INH+RIF x 4 months



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 → malignancy

rapid growth → infection

no growth → 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		
	<i>malignant</i>	<i>benign</i>
<i>age</i>	> 45-50	< 35
<i>calcifications</i>	absent to irregular calcifications	central/uniform/laminated or popcorn like
<i>size</i>	>2cm	<2cm
<i>old films</i>	new or larger	no change
<i>margins</i>	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 than 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

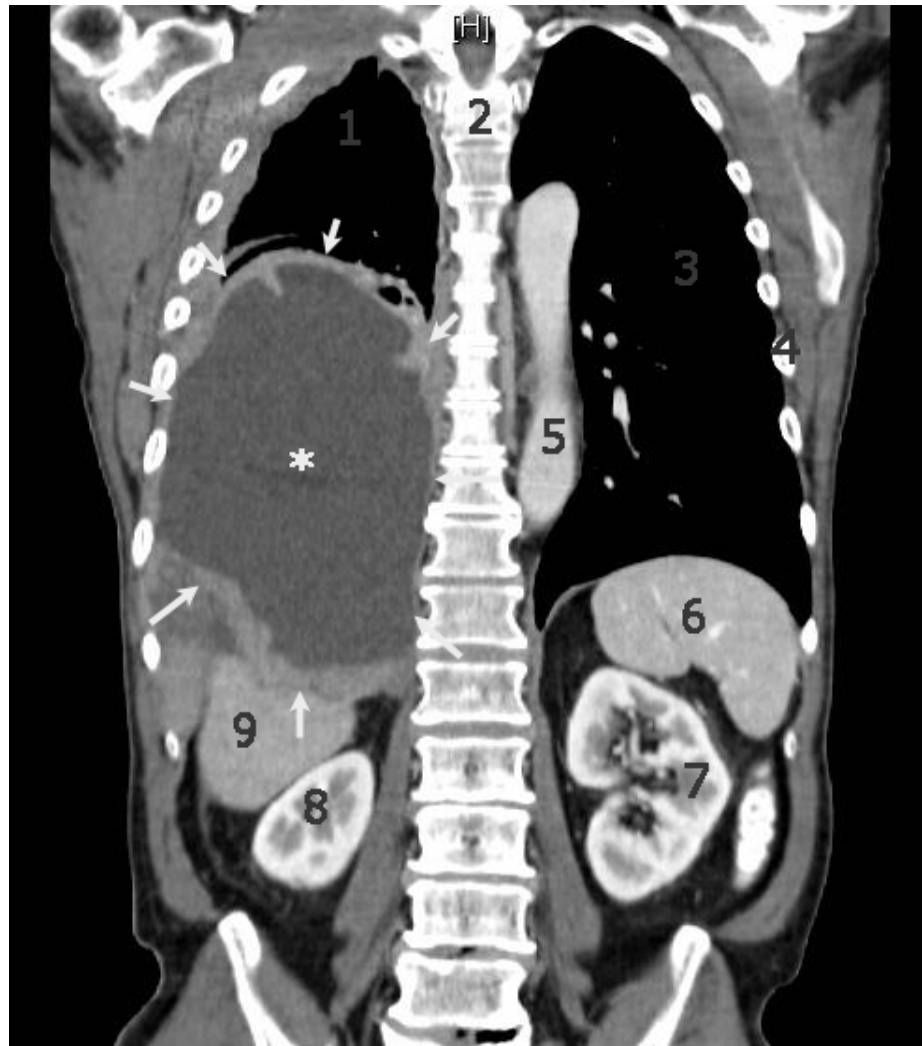
video assisted thoracic surgery (VATS): biopsy

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 mets to lung:

breast, liver, colon

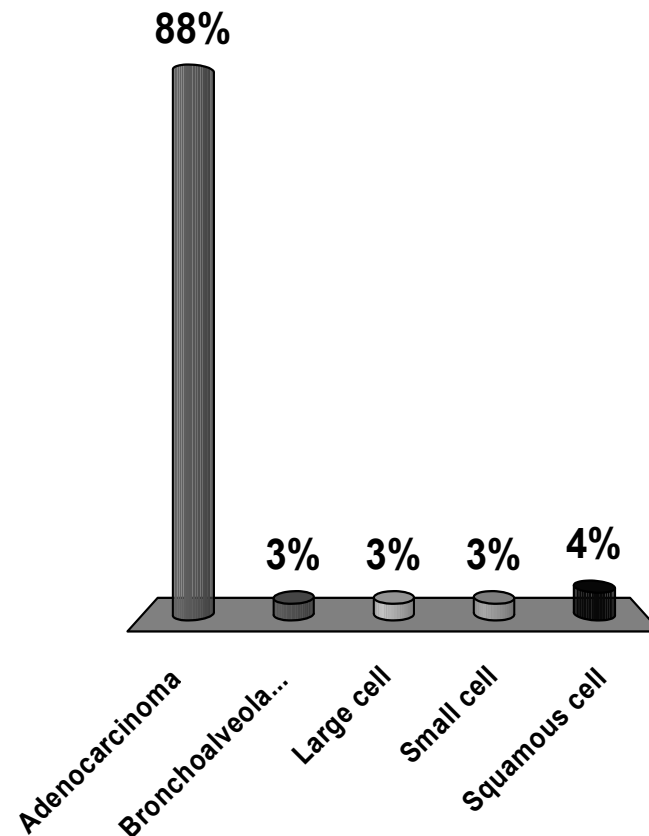
almost any CA can spread to lungs

imaging reveals multiple nodules/masses

diagnose and treat the primary tumor

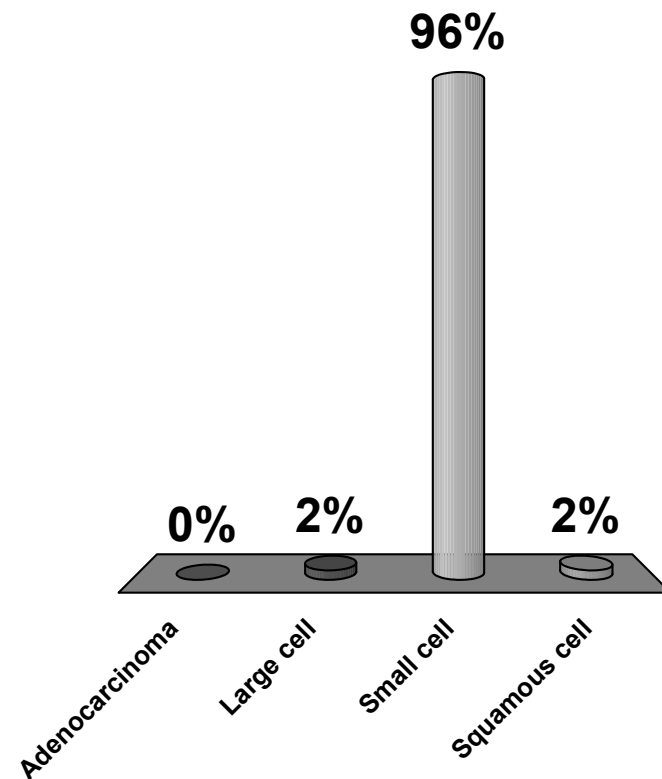
Which of the following is the most common type of lung cancer in non-smokers?

- A. Adenocarcinoma
- B. Bronchoalveolar
- C. Large cell
- D. Small cell
- E. Squamous cell



For which of the following types of lung cancer is surgery generally not indicated?

- A. Adenocarcinoma
- B. Large cell
- C. Small cell
- D. Squamous cell



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	$\leq 2x/\text{week}$	$\leq 2x/\text{month}$	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: metacholine 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

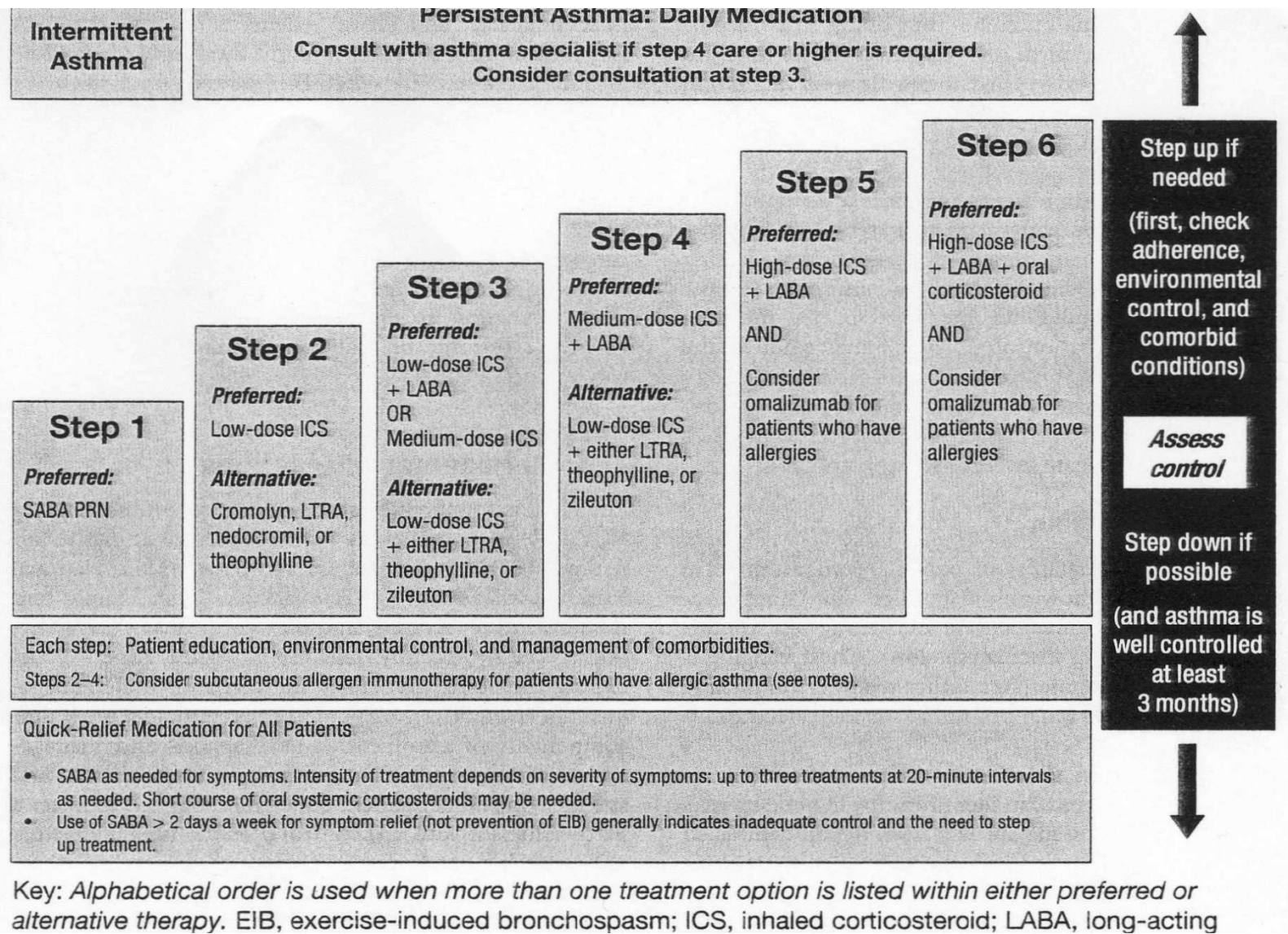
INH mast cell stabilizers (e.g. cromolyn)

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):

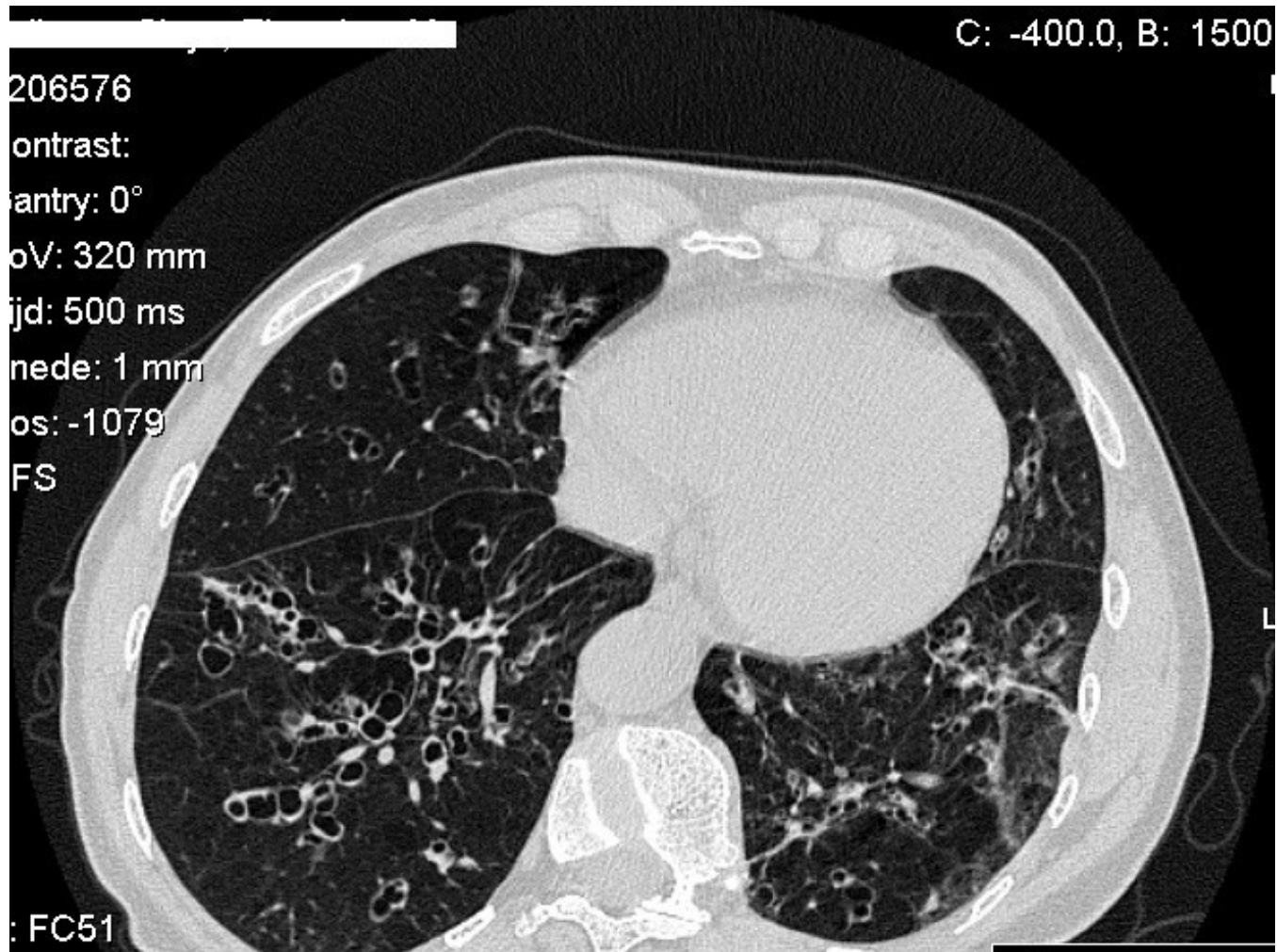
amoxicillin

Augmentin

Bactrim (TMP/SMX)

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](https://pubmed.ncbi.nlm.nih.gov/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= }}
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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	<p><i>Hallmark: exertional dyspnea</i></p> <p>cough is rare</p> <p>quiet lungs</p> <p>no peripheral edema</p> <p>thin; recent weight loss</p> <p>barrel chest</p> <p>pursed lips breathing</p> <p>hyperventilation</p>	<p>mild dyspnea</p> <p><i>chronic productive cough</i></p> <p>noisy lungs: rhonchi and wheeze</p> <p>peripheral edema</p> <p>overweight and cyanotic</p>
CXR	<p>decreased lung markings at apices</p> <p>flattened diaphragms</p> <p>hyperinflation</p> <p><i>parenchymal bullae and blebs</i></p> <p>small, thin appearing heart</p>	<p>increased interstitial markings at bases</p> <p>diaphragms not flattened</p>



CXR of patient with severe emphysema.

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

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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

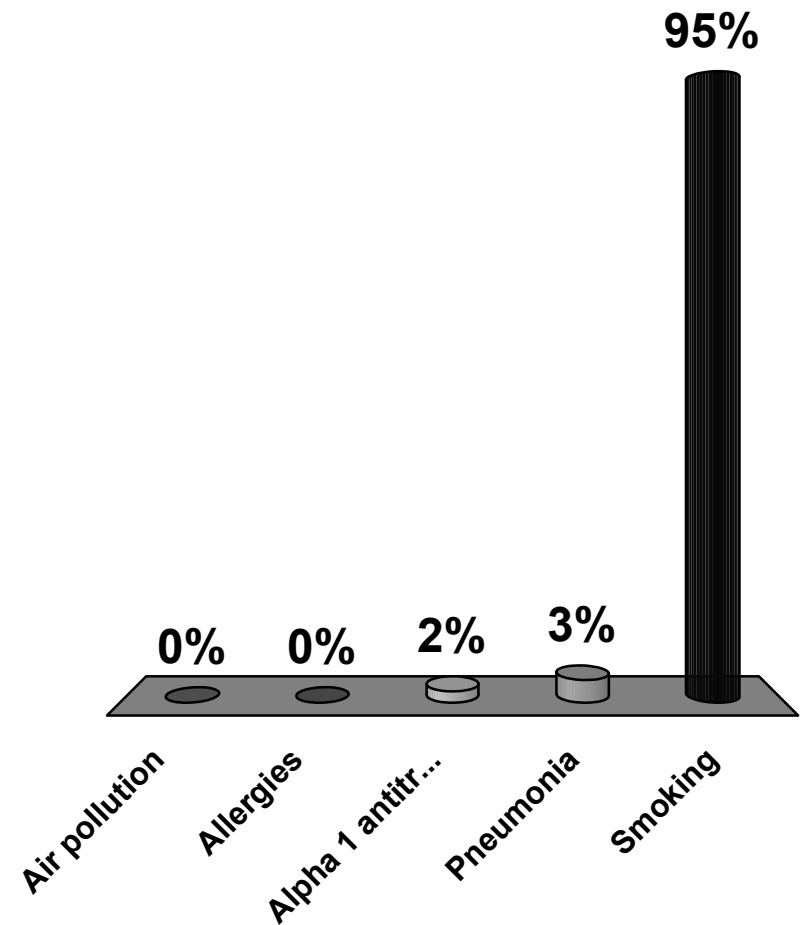
TMP/SMX; augmentin/clavulanate,
doxycycline

influenza and pneumococcal vaccines

Surgery: transplant, LVRS, bullectomy

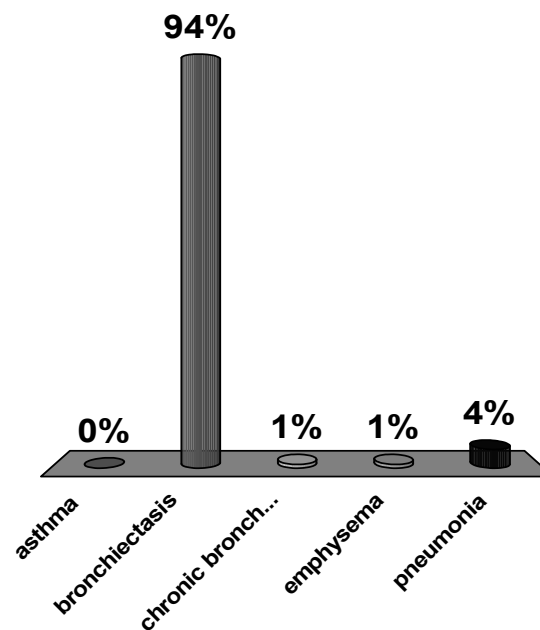
Which of the following is the most common cause of chronic bronchitis?

- A. Air pollution
- B. Allergies
- C. Alpha 1 antitrypsin deficiency
- D. Pneumonia
- E. Smoking



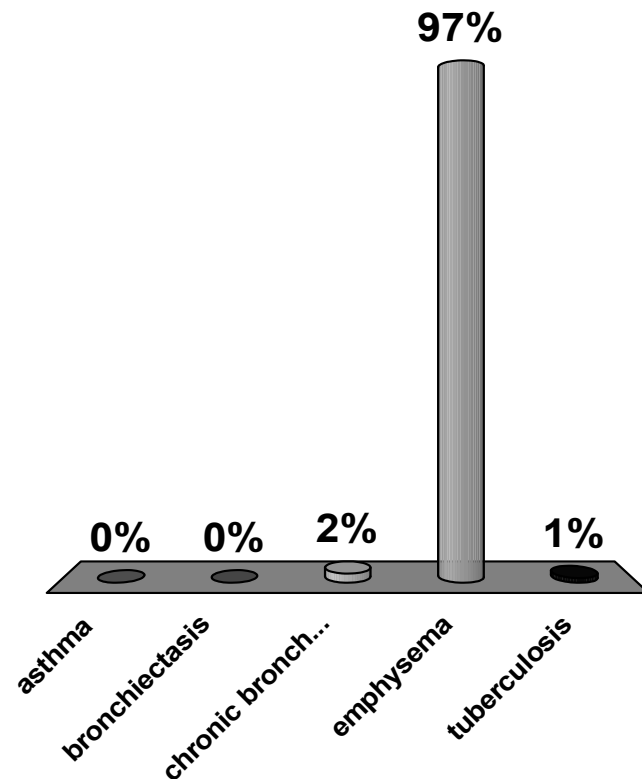
A 12 year-old girl with cystic fibrosis since age 3 presents c/o purulent foul smelling sputum. CXR demonstrates tubular, air-filled structures that extend to near the end of the lung fields. The remainder of the lung fields appear normal. Which of the following is the most likely diagnosis?

- A. asthma
- B. bronchiectasis
- C. chronic bronchitis
- D. emphysema
- E. pneumonia



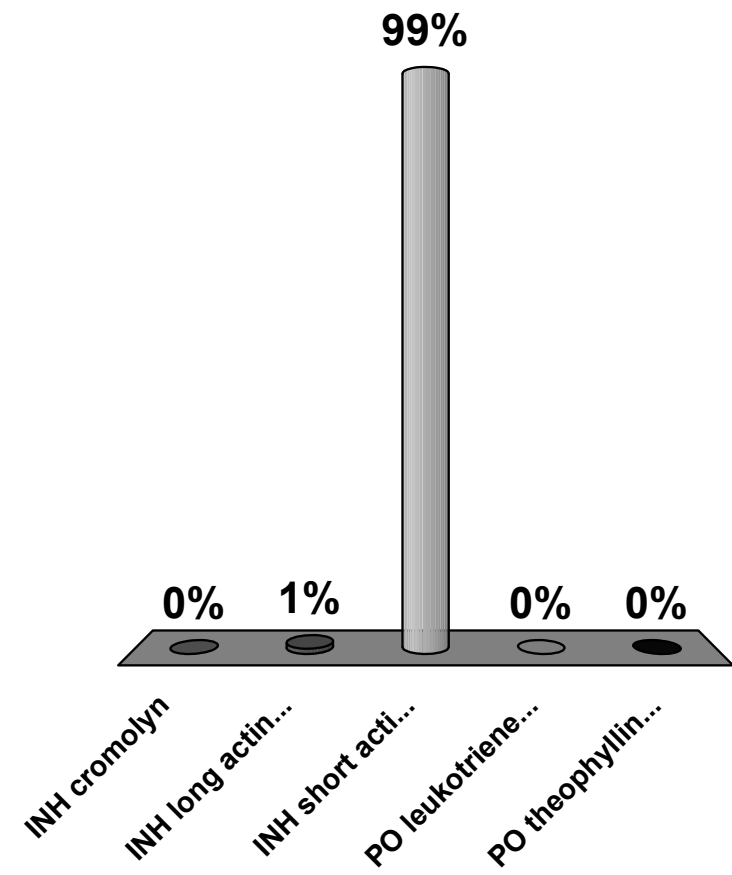
A 60 year-old male smoker presents c/o SOB with mild exertion. He denies cough or chest pain. Exam reveals a thin male with an increased chest A:P diameter and use of accessory muscles. On auscultation, the chest is very quiet with no adventitious sounds. Which of the following is the most likely diagnosis?

- A. asthma
- B. bronchiectasis
- C. chronic bronchitis
- D. emphysema
- E. tuberculosis



A 24 year old woman is having an acute asthma exacerbation. Which of the following medications would be most appropriate to administer?

- A. INH cromolyn
- B. INH long acting beta-2 agonist
- C. INH short acting beta-2 agonist
- D. PO leukotriene modifier
- E. PO theophylline



Part IV:

Restrictive Pulmonary Diseases

- Idiopathic Pulmonary Fibrosis
- Pneumoconioses
- Sarcoidosis

Restrictive Pulmonary Diseases

Normal / \uparrow FEV/FVC

\downarrow TLC

Idiopathic Pulmonary Fibrosis

Background:

most common dx among pts with interstitial lung disease
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

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 <i>mesothelioma</i> , 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 <i>TB</i> ; progressive massive fibrosis
berylliosis	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 non-caseating granulomas

Treatment: prednisone

Other: “**GRUELING**”

Granulomas

RA

Uveitis

Erythema nodosum

Lymphadenopathy

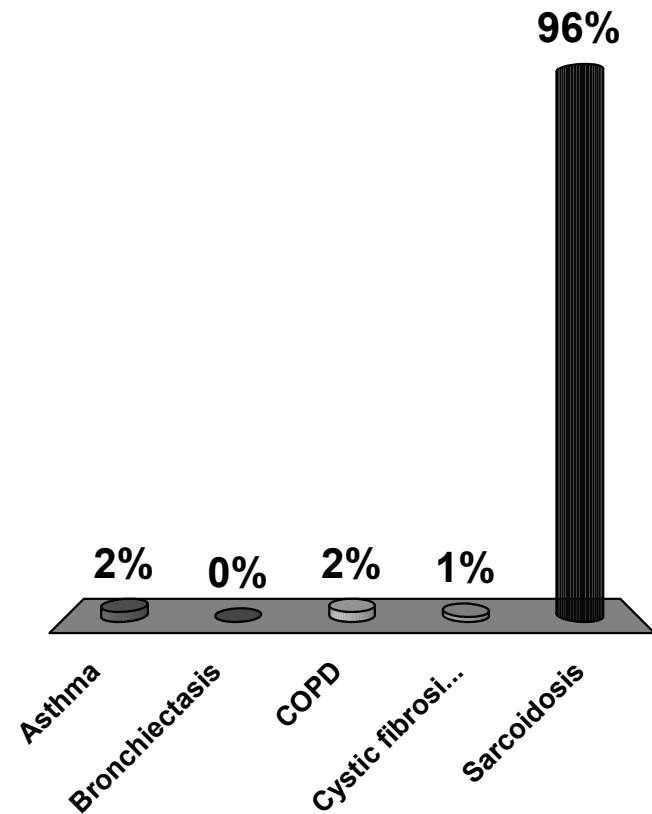
Interstitial fibrosis

Negative TB test

Gammaglobulinemia

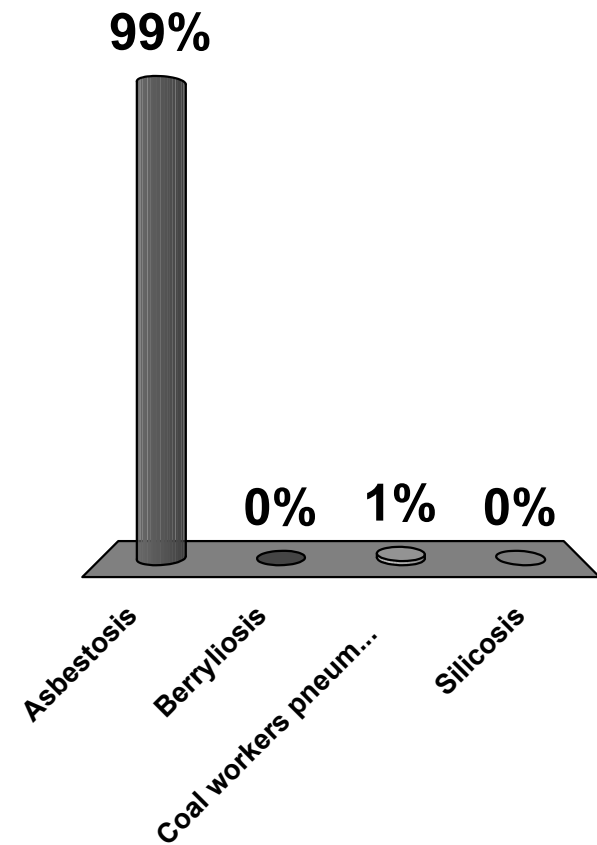
Which of the following is an example of a restrictive lung disease?

- A. Asthma
- B. Bronchiectasis
- C. COPD
- D. Cystic fibrosis
- E. Sarcoidosis



Which of the following restrictive lung diseases has an increased risk of mesothelioma if the patient is a smoker?

1. **Asbestosis**
2. Berylliosis
3. Coal workers pneumoconiosis
4. Silicosis



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

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)

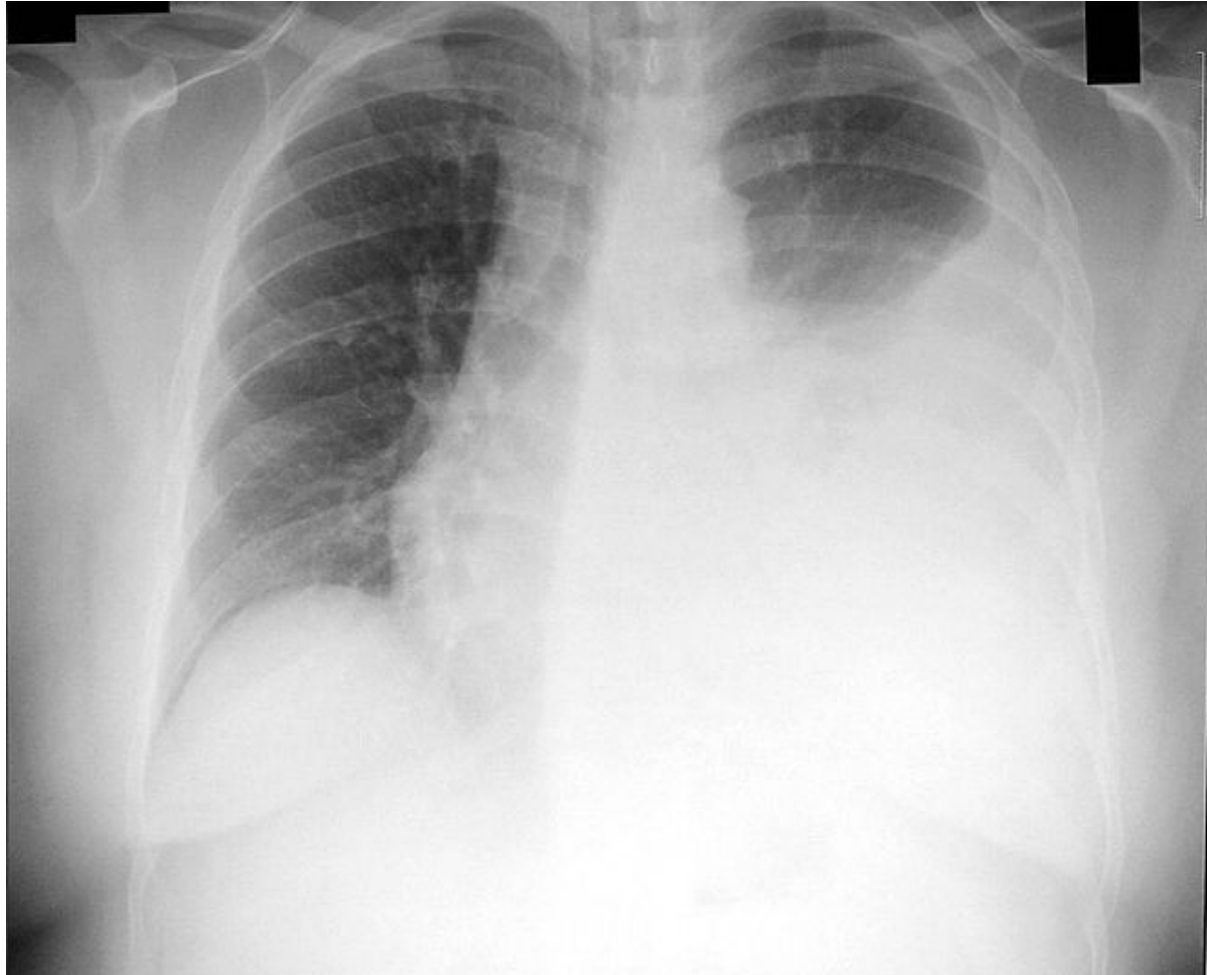
CT scan for small effusions

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

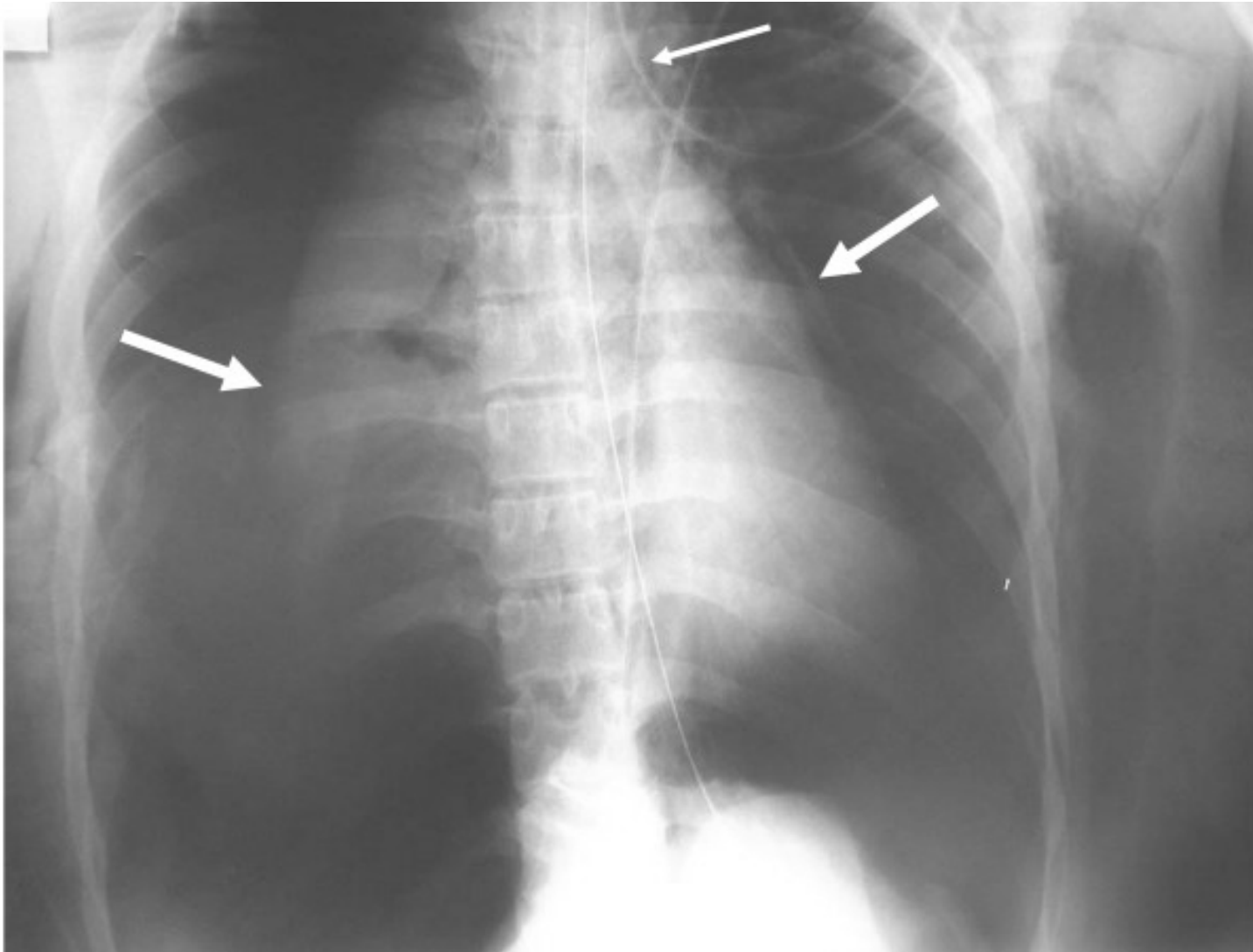
Treatment:

Primary spontaneous/secondary:

<15% diameter of hemithorax on CXR: rest, cough,
chest pain relief, serial CXRs

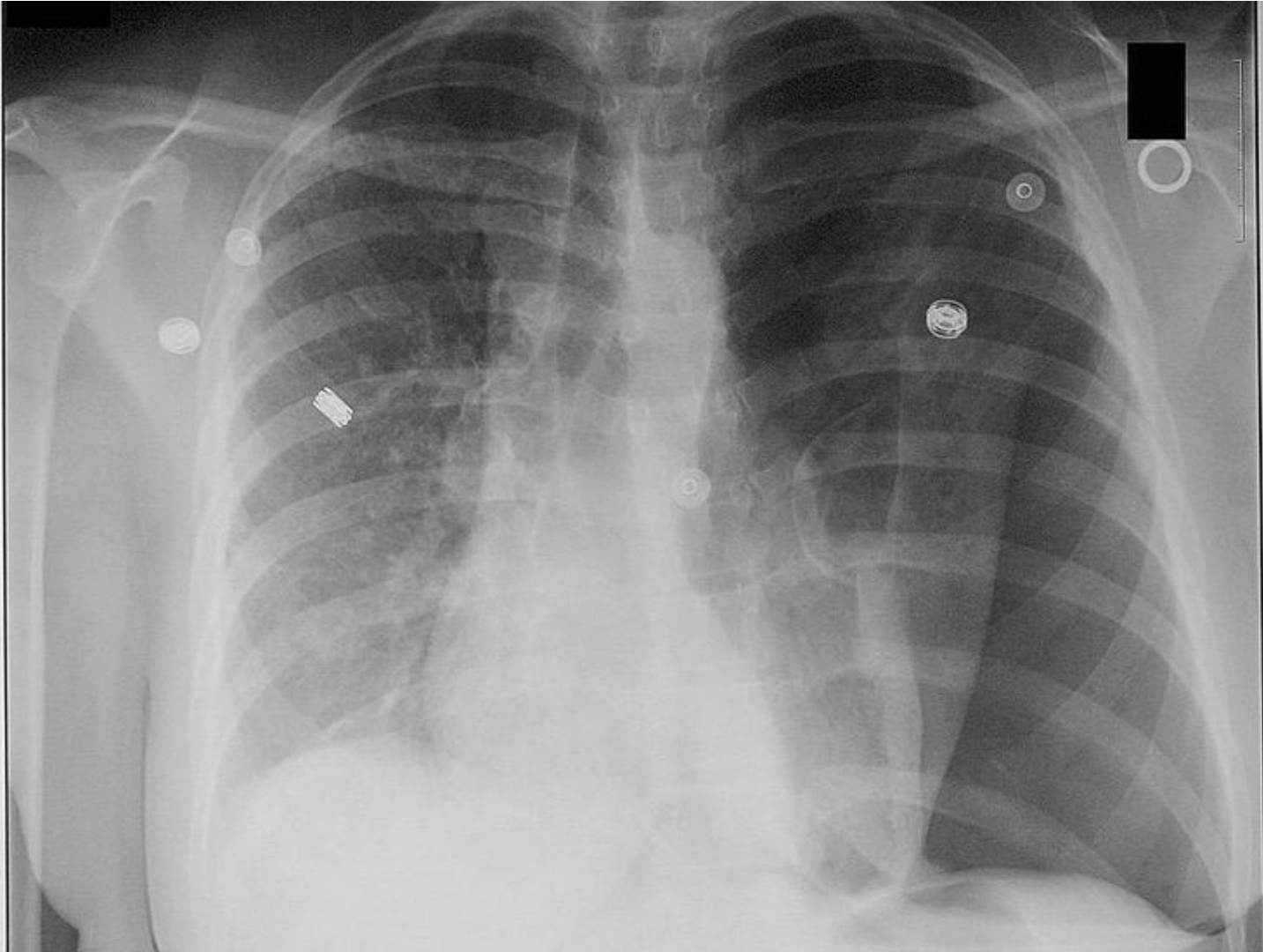
>15%: chest tube plus above measures

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>

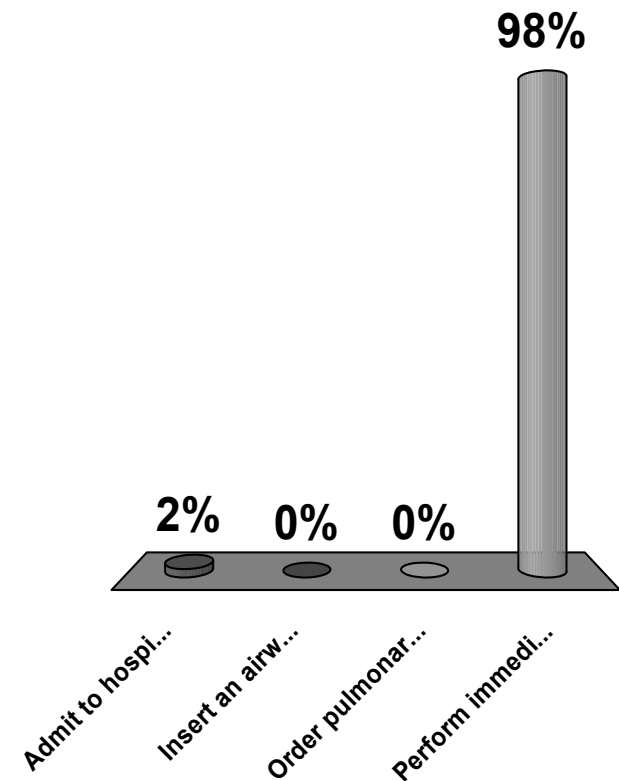


Left tension pneumothorax

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

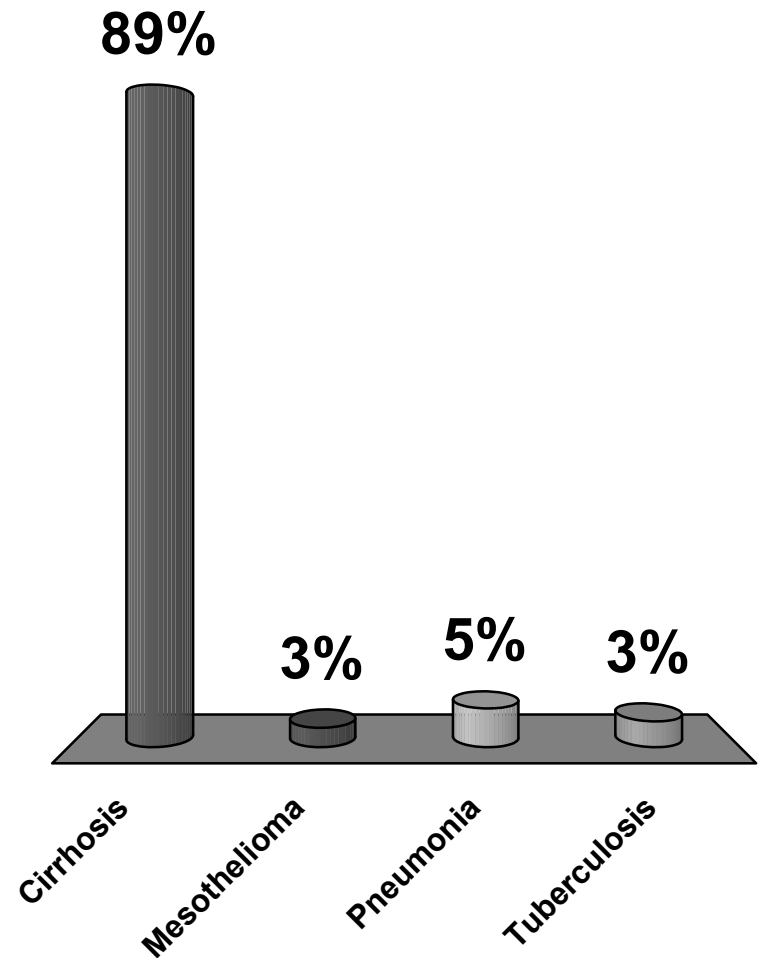
A 54 year-old patient presents with acute onset of chest pain, SOB and hypotension. CXR reveals a mediastinal shift to the right. Which of the following is the most appropriate next step?

- A. Admit to hospital for observation
- B. Insert an airway
- C. Order pulmonary consult
- D. Perform immediate needle decompression



Which of the following is the most likely cause of an transudative effusion?

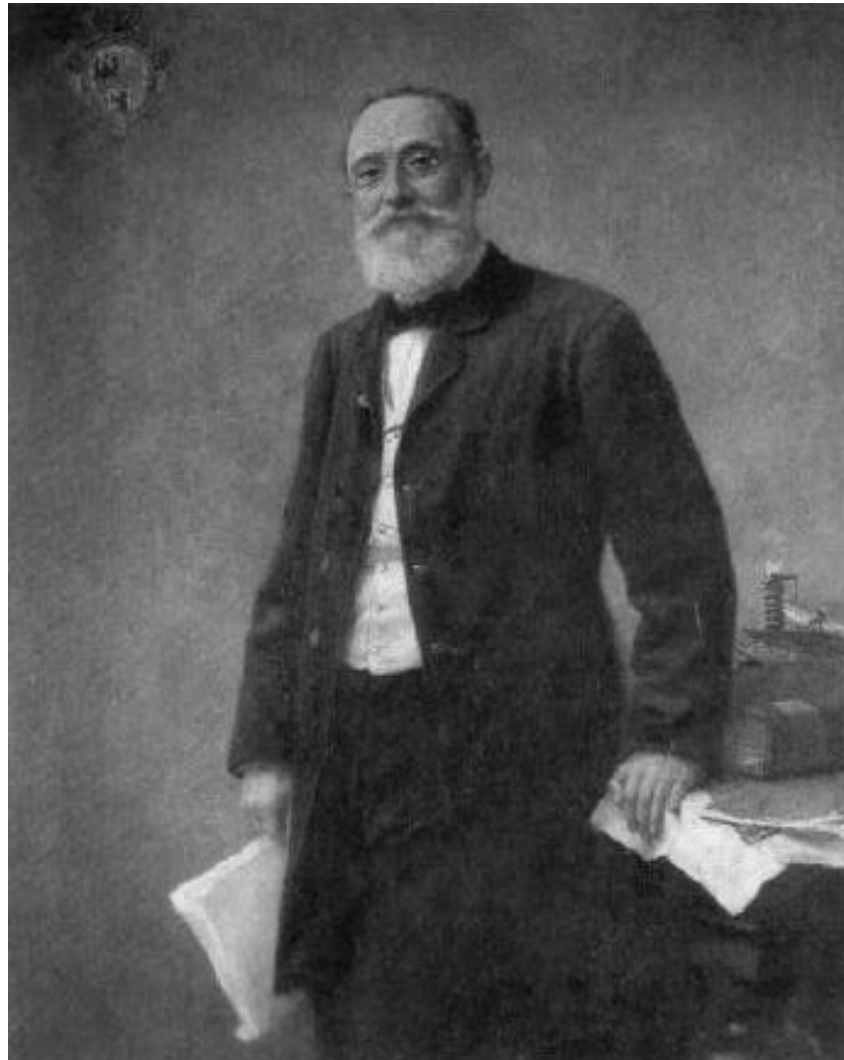
- A. Cirrhosis
- B. Mesothelioma
- C. Pneumonia
- D. Tuberculosis



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>

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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 patient with 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

Westermarck'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

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Pulmonary Embolism (PE)

Spiral CT (helical) angiography

more sensitive than 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):
only for stable patient

not generally recommended

IVC filter

surgery: only for saddle emboli

Pulmonary Embolism (PE)

Other:

Prevention:

combination of mechanical & pharmacological
measures

early ambulation

intermittent pneumatic compression

low dose heparin

LMWH

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

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 respiratory 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 $> 30^\circ$; flat, inverted T waves in RV precordial leads

Treatment:

oxygen

decrease PVR 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)

$\text{PaO}_2/\text{FiO}_2 < 200$

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%

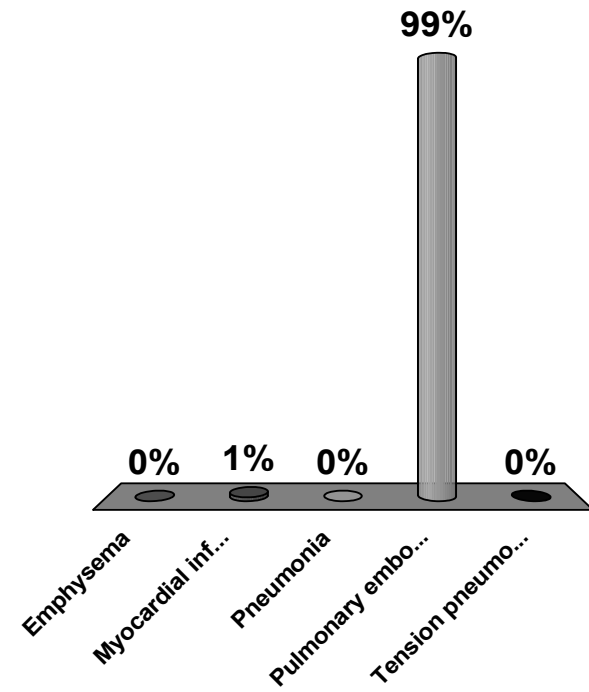
Other:

ARDS mortality: 30-40%

ARDS *plus* sepsis mortality: 90%

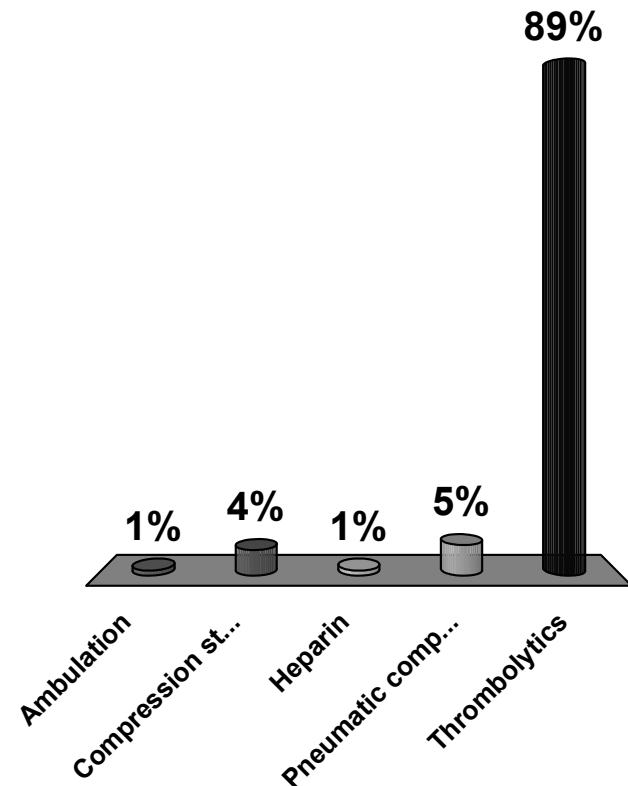
A 52 year-old patient presents to the ER with pleuritic chest pain, cough, dyspnea, and hemoptysis. On exam she is anxious with tachycardia and tachypnea. Lab work demonstrates and elevated D-dimer. CXR is normal. What is the most likely diagnosis?

- A. Emphysema
- B. Myocardial infarction
- C. Pneumonia
- D. Pulmonary embolism
- E. Tension pneumothorax



Which of the following is not recommended for primary prevention of pulmonary emboli in the immediate post-op period?

- A. Ambulation
- B. Compression stockings
- C. Heparin
- D. Pneumatic compression
- E. **Thrombolytics**



Good luck!