

General

- Normal Pleural Fluid: ~10cc, clear/straw colored, nonviscous, low protein, <1k cells /mCL predominantly macrophages/mesothelial cells/monocytes, pH 7.60-7.65 (note not ~serum)
- Parietal Pleura
 - Lines the thoracic cage, mediastinum, and diaphragm
 - Supplies pleural fluid via intercostals arteries close to surface hence creates majority of fluid
 - Does NOT remove pleural fluid
- Visceral Pleura
 - Surrounds the lungs
 - Supplies pleural fluid via bronchial arteries far from surface hence creates minority of fluid
 - Removes pleural fluid via pulmonary veins and lymphatics (aided by negative pleural pressure)
- NB the right and left pleural spaces are completely separated by the mediastinum
- NB there is a constant flux of pleural fluid from parietal to visceral based on removal of fluid
- NB pleural fluid is generated by systemic circulation and removed by pulmonary circulation
- NB lymphatic drainage is complicated and not completely understood but one very important feature is known: though venous drainage is the primary drainage route for pleural fluid lymphatic drainage is able to increase efficacy in response to pleural effusions
- NB the peritoneal cavity can also remove fluid from the right side

Pneumothorax (PTX)

- Types
 - **Spontaneous**
 - **Primary (NO overt lung dz):** 2/2 unprovoked rupture of congenital small apical subpleural blebs in otherwise normal lungs in tall lean young men who smoke, 50% recurrence in 2yrs unless smoking is stopped, these pts have good pulmonary reserve so Sx are rare or minimal
 - **Secondary (overt lung dz):** 2/2 rupture of alveoli due to: obstructive pulmonary disease (COPD, Asthma), infections (TB, Pneumocystis carinii, necrotizing bacterial, anaerobic, Staph), malignancy (sarcoma, lung), necrotizing granulomatous processes (eosinophilic granuloma, sarcoid), lymphangioleiomyomatosis (young women), pulmonary infarction
 - **Non-Spontaneous**
 - **Iatrogenic:** 2/2 MV barotrauma, thoracentesis, central line placement, bronchial biopsy, etc
 - **Traumatic:** 2/2 perforating injury to the chest wall
- Tx: no Sx (SOB, ipsilateral cp, cough w/ decreased BS, hyperresonance, decreased fremitus, mediastinal/tracheal shift TOWARD side of PTX) and <50yo and < ½ hemithorax and Iatrogenic/Primary then observe b/c will likely resorb or needle aspiration otherwise chest tube (don't place to suction b/c of re-expansion pulmonary edema unless persistent >48hrs) and then consider pleurodesis after re-expansion
 - NB Tension (usually occurs in MV/traumatic causes, so severe that air compresses lung parenchyma in such a way that it acts as a flap valve permitting entrance of air during inspiration but NOT escape of air during expiration with the net effect of a quick increase in air resulting in compression of venous return, mediastinal/tracheal shift AWAY FROM side of PTX, lung collapse and depression of ipsilateral diaphragm ultimately leading to severe hypoxemia) then emergent needle decompression, oxygen, chest tube

Pleural Effusion

- Diagnosis
 - Evaluate Size of Effusion
 - CXR AP/Lateral: it takes >250cc of fluid to visualize blunting of costophrenic angles
 - CXR Lateral Decubitus: once visualized on AP/lateral determine if free-flowing vs loculated and whether enough fluid is present (>10mm) that thoracentesis can safely be performed at bedside otherwise call radiologist to see if it can be done under US guidance by IR
 - CT Chest is good CXR is equivocal b/c differentiating degree of loculations, masses vs fluid, atelectasis vs fluid (some always order a CT it just helps if there is ever confusion)
 - US can also be helpful
 - Differentiate Transudative vs Exudative Process by looking at Light's Criteria b/t serum and effusion via thoracentesis
 - NB Light's Criteria: high sensitivity (~100%) and low specificity (~80%) (depending on the number of Light's Criteria met 1-3/3) therefore some transudative pts will be misclassified as exudative pts, this is most commonly seen in CHF if chronic and/or diuretic use (more liquid than protein is removed), therefore if CHF or you clinically suspect transudative but you get you get exudative results then check Albumin Gradient, Cholesterol in Effusion, Cell Count, etc b/c some conditions may have high small proteins but no way in hell albumin (a big protein) or cholesterol or actual cells

"Light's Criteria" for Exudative Process						
	$\text{LDH}_{\text{effusion}}$ (mg/dL)	$\frac{\text{LDH}_{\text{effusion}}}{\text{LDH}_{\text{serum}}}$	$\frac{\text{TP}_{\text{effusion}}}{\text{TP}_{\text{serum}}}$	$\text{Alb}_{\text{serum}} - \text{Alb}_{\text{effusion}}$ (mg/dL)	Chol_{eff} (mg/dL)	Cell Count
Transudate Low Protein	<2/3ULN $\text{LDH}_{\text{serum}}$	<0.6	<0.5	>1.2	<45	WBC<1k RBC<10k

Low Cell						
Exudate High Protein High Cell	>2/3ULN LDH _{serum}	>0.6	>0.5	<1.2	>45	WBC>1k RBC>10k

- If clinical suspicion is exudative and fluid is exudative by Light's Criteria then look at cell counts, glucose/pH and other labs
 - Cell Counts:
 - Glucose: Serum ~ Trans ~ Exudative but some Exudative have low glucose which is diagnostically and prognostically helpful b/c the more acidotic the higher M&M especially for infection and malignancy
 - pH: Serum 7.35-7.45, NI Eff 7.60-7.65, Trans 7.45-7.55, Exu 7.30-7.45 but some exudative are very acidotic which " "
- S/S: asymp, local/referred to shoulder/general pleurisy, dyspnea, cough
- Tx:
 - Transudative Process
 - Treat Underlying Problem
 - Diuretics and Sodium Restriction
 - Therapeutic Thoracentesis
 - If effusion requires recurrent thoracentesis and pt is symptomatic and gets better after thoracentesis then consider
 - subacute pigtail catheter and then chronic indwelling pleural catheter (Pleurx catheter)
 - pleurodesis w/ Talc (effective and inexpensive but fever, hypoxia, and rarely respiratory failure), Doxycycline/Minocycline (effective and inexpensive but pain), Bleomycin (less effective and expensive)
 - pleurectomy
 - pleural abrasion
 - pleuralperitoneal shunting
 - Exudative Process
 - Treat Underlying Problem
 - Refer Below
- Etiology
 - **Transudative Process** (30% CHF, 30% Cirrhosis, 20% **Undiagnosed** therefore evaluate for chronic asymptomatic PE)
 - Mechanism
 - Increased drainage into pleural space
 - Increased Hydrostatic Pressure on visceral or parietal pleura
 - Decreased Oncotic Pressure on visceral or parietal pleura
 - Decreased drainage out of pleural space
 - **CHF** (can be psuedoexudative if chronic or diuretic use, 15% unilateral, can diurese first as 75% resolve in 2d but if you have other concerning Sx suggesting possibly another etiology then go ahead w/ thoracentesis)
 - **Cirrhosis aka Hepatic Hydrothorax** (2/2 to low albumin and/or direct extension of ascites into right pleural cavity thru small diaphragmatic defects, 15% bilateral, can diurese first)
 - **Nephrotic Syndrome** (usually small, bilateral, asymp but if a pt presents with symptomatic effusions consider PE b/c of the hypercoagulable state of nephrotic syndrome)
 - **Hypoalbuminemia** (2/2 above and malnourished state, etc)
 - **Pulmonary Embolus w/o Infarction**
 - **Iatrogenic** (2/2 vascular erosion by central lines)
 - **Trapped Lung** (after a mild asymptomatic inflammatory process (infection, CTD, etc) a fibrous membrane can develop on visceral pleura preventing lung expansion creating negative pressure sucking fluid into space)
 - **Urinothorax** (very rare, occurs when there is obstructive uropathy when urine exudes from kidney capsule and moves retroperitoneally into pleural space, fluid ~urine, effusion Cr > serum Cr)
 - **Peritoneal Dialysis** (can be chronic or rarely acute after initiation of PD, very high glucose)
 - **Atelectasis** (esp post-op b/c of left diaphragmatic dysfunction 2/2 phrenic nerve injury 2/2 MV)
 - **Exudative Process** (30% Infection, 30% Malignant, 20% **Undiagnosed** therefore evaluate for PE, TB and unusual ab dz like splenic infarct, ovarian tumor, etc)
 - Mechanism
 - Increased drainage into pleural space
 - Increased Permeability of visceral or parietal pleura
 - Increased production from pleural space
 - **Most Important/Serious/Common Ones signified by the fact that Glu is <serum and pH <7.3 hence rule out and treat first**
 - **Infection: Pneumonia aka Parapneumonic Effusion, TB, Hepatic/Splenic Abscess, etc**
 - Serous (uncomplicated) → Empyema (complicated) → Organization w/ Fibrosis and formation of rigid pleural peel
 - Variable WBC
 - >50% Neutrophils (PNA, PE, pancreatitis)

- >50% Lymphs (TB, Cancer)
 - >10% Eos (refer below)
 - Always inoculate into blood culture tubes at bedside (aerobe, anaerobe, fungal)
 - Uncomplicated (aka non-infected) vs Complicated (aka infected indicated by + Gram Stain OR + Cx OR pH <7.2 OR glu <serum OR LDH >600)
 - pH: quickly send to blood gas lab on ice, only check if you suspect parapneumonic/malignant effusion, <7.20 infection = chest tube, <7.30 cancer = decreased survival
 - TB: most common extra-pulm TB, 2/2 subpleural TB infection ruptures, check AFB Stain (10% sens), Cx (40% sens), Pleural Bx (70%), PCR (100%) and ADA (Adenosine DeAminase), enzyme used in degrading purines in every cell) >70 w/ <40 excluding TB
 - Tx: abx unless complicated or empyema or >1/2 hemithorax or air-fluid levels or loculations (collectively represents 15% of infectious effusions hence most resolve w/ abx alone) then chest tube, if loculated or persistent sepsis despite above then you will need thrombolytics or LOA via VATS/thoracotomy, if organization does occur then decortifications
- **Malignancy w/ mets to pleura w/ or w/o lymph obstruction**
 - Primary: **Benign Asbestos Pleural Effusion (BAPE)** (benign effusion w/o any cancer benign/malignant), **Benign Solitary Fibrous Tumor** (CD34+/Keratin-, whorls of collagen and reticulin with spindle shaped cells), **Malignant Mesothelioma** (CD34-/Keratin+, asbestos bodies and plaques, 2/2 asbestosis exposure usually decades ago w/ 30yr latency, NB concurrent pulmonary asbestosis is only in 20% of pts, metastasizes to first lungs then to hilar LNs then liver, 50% die within first 1 year, Tx w/ Carboplatin (Paraplatin) + pemetrexed (Alimta), Dx: Osteopontin
 - Secondary: metastatic tumor from **1° lung/breast, lymphoma, stomach, ovary**, can have high amylase so if no esophageal perforation or pancreatitis then very likely cancer, cytology but only + 50/60/70% w/ 1st/2nd/3rd sample, (variable depending on type of cancer (70% adeno vs <25% all others) and b/c most malignant effusions are paramalignant in which cancer has not directly metastasized to pleura but still causes effusions for other reasons specifically production and circulation of VEGF, obstruction of lymphatics/bronchi, PE, 2/2 Tx w/ chemo/radiation, etc) therefore if negative and suspicious then repeat for a total of 3 samples and if negative again and still suspicious then Thoracoscopic Bx, Tx: based on specific cancer type, pleurectomy
 - Typically few WBC and variable RBC
 - Others
 - **Connective Tissue Disease** (can have glu<serum and pH<7.3, + ANA/RF esp if > serum, you can have unusual cytology in RA)
 - **PE w/ Infarction:** WBC <50k, neutrophilic, some RBC, unilateral, AC is not contraindicated even if there are RBCs is fluid)
 - **Radiation** (usually done for Tx cancer therefore must r/o cancer as the cause of the effusion)
 - **HypoTH** (actually rare so if must exclude other causes before you can see an effusion is 2/2 hypoTH)
 - **Yellow Nail Syndrome aka YNS** (absence of certain lymphatics → lymphedema + yellow nails + respiratory tract infections w/ pneumonia, bronchitis, sinusitis, bronchiectasis and lymph predominant pleural effusions)
 - **Meig's Syndrome** (ovarian tumor + ascites + pleural effusion)
 - **Uremia** (different than urinothorax, 2/2 vascular injury, effusion<serum Cr)
 - **Sarcoidosis** (lymphs)
 - **Post-Cardiac Injury Syndrome** aka PCIS (~3wks following any type of cardiac surgery or ACS, 2/2 immune response to injured epi/myocardium, +anti-myocardial antibodies, high APRs left sided, serosanguinous w/ neutrophils then lymphocytes)
 - NB
 - **Chylothorax**
 - TGL <50: psuedochylothorax
 - TGL 50-110: check lipoprotein electrophoresis for presence of chylomicrons b/c if present then b/c if + then refer below
 - TGL >100: chylothorax (2/2 Thoracic Duct Damage (CT-surgery) / Compression (cancer) / Back-Up (left SC vein thrombosis), LymphAngioleiomyomatosis aka LAM (smooth muscle proliferation around airways/vessels/lymphatics, complicated by PTX/hemoptysis/chylothorax respectively, seen in young women 2/2 estrogen effects), Lymphoma, Idiopathic, etc, left sided, milky effusion
 - **Hemothorax**
 - Hct <1% of serum: not significant

- Hct 1-20% of serum: cancer and PE, coagulopathy, leaking aortic aneurysm, aortic dissection
 - Hct >20% of serum: hemothorax
- **Eosinophils:** 1° pnemothorax, 2° hemothorax, 3° BAPE, PE, prior thoracentesis, parasite/fungal infection, drugs induced (dantrolene, bromocryptine, nitrofurantoin, amio), lymphoma, cancer
- **Amylase: Pancreatitis** (increased permeability 2/2 inflammation and as amylase collects in effusion it is protected from being cleared by kidney hence amylase effusion>serum), **Esophageal Endoscopy w/ or w/o Tx resulting in Perforation or Vomiting/Retching leading to Boerhaave's Syndrome** (left sided, glu <serum/pH<7.3, anaerobic infection often results, squamous epi cells and food particles), **Malignancy**
- **Chronic Effusions Lasting >1yr:** Yellow Nail Syndrome, Trapped Lung, Lymphangioleiomyomatosis, Chylothorax



Copyright 2015 - Alexander Mantas MD PA