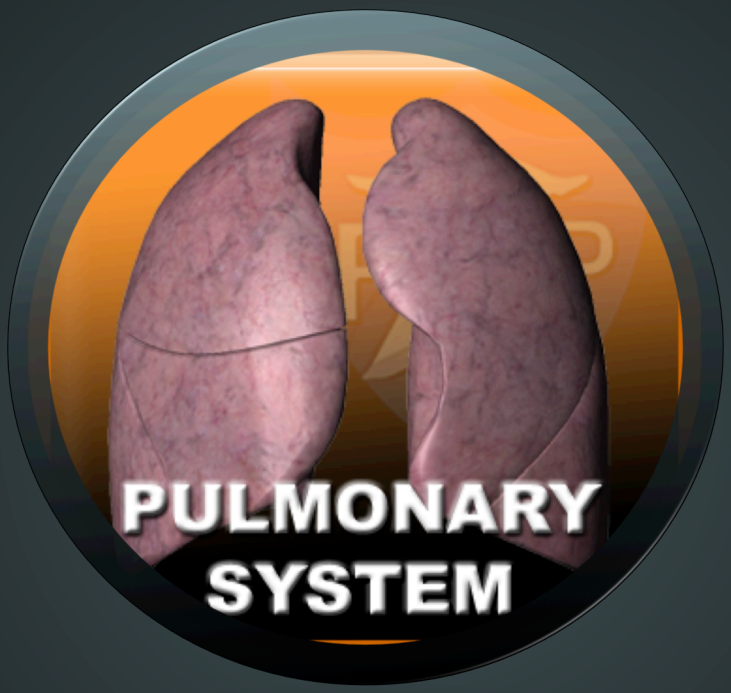
Ftplectures Pulmonary system Lecture Notes

PULMONARY



Medicine made simple

Ftplectures Clinical Medicine
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The field of Medicine is an ever-changing profession and as new evidence based studies are conducted, new knowledge is discovered. Ftplectures has made tremendous effort to deliver accurate information as per standard teaching of medical information at the time of this publication. However, there are still possibilities of human error or changes in medical sciences contained herein. Therefore, ftplectures is not responsible for any inaccuracies or omissions noted in this publication. Readers are encouraged to confirm the information contained herein with other sources.

ACUTE RESPIRATORY FAILURE (ARF)

Title: Abdominal Aortic Aneurysm

Objectives For Learning: Key Definitions, Causes/ Risk factors, Classification Of Acute Respiratory Failure, Clinical symptoms and signs, Diagnosis, Treatment and Complications.

Definition:

Hypoxia: Decrease oxygen to the tissues
Hypoxemia: It is the low oxygen dissolved in the plasma.

Oxygen saturation: It is the percentage of oxygen binds to the hemoglobin.
Calculating A + a gradient
PAO_2 - P_AO_2

PAO_2= % (760 – PH_2O)- $PaCO_2$

Normal P_AO2 = 100 mmHg

Normal P_AO_2 = 40mmHg

Causes/ Risk factors:

1. CNS:

Brain trauma
Drug overdose
Stroke

2. Anything that can affect spinal cord
3. Neuromuscular disease
   - Myasthenia gravis
   - Polio
   - Guillain–Barré syndrome
   - Amyotrophic lateral sclerosis
4. Upper airways
   - Upper airway stenosis
   - Paralysis of vocal cords
   - Hemothorax
   - Kyphoscoliosis
   - Flail chest
5. Cardiovascular system
   - Heart failure
   - Pulmonary embolism
6. Lower Airway Diseases

- COPD
- Asthma

Classification Of Acute Respiratory Failure

There are two types

1. **Hypoxemic Respiratory Failure:** Three things are needed to know
   - $PaCO_2$
   - A-a gradient
   - Response to supplemental oxygen

Causes of hypoxemia

1. V/Q defect

V= ventilation
Q= perfusion

2. Pneumonia
3. ARDS
4. Pulmonary edema

2. Hypercapnic Or Ventilatory Respiratory Failure

Increased $PaCO_2$

- It is the ventilatory respiratory failure.

$V_A = RR - V_T$

V_A = Alveolar ventilation
$V_{T=}$ Tidal Volume

This is when too much carbon dioxide is retained within the body. Eventually hypoxemia develops secondary to hypercapnia. This is seen in the following conditions.

- Patients that have respiratory depression or CNS problems
- COPD
- Asthma
- Prolonged hyperventilation because of diabetic ketoacidosis.

The ratio of PO_2 to FIO2 is always less or equal to 200. They always have diffuse bilateral infiltration of both lungs and pulmonary capillary wedge pressure is less than 18.

Clinical Symptoms And Signs

The most common symptom is dyspnea. – it is the first symptoms of acute respiratory failure. Other includes Cough

Signs:

- Use of accessory muscles

- Talking incomplete sentences
- Tachpnea
- Tachycardia
- Impaired ventilation

Diagnosis

1. Arterial blood gas to measure $PaCO_2$ and PaO_2
2. Blood pH
3. Chest X-ray
4. CT scan chest
5. CBC
6. Troponin level to evaluate myocardial infraction

Treatment

- Treat the cause. Give bronchodilators, Corticosteroids, antibiotics, etc, depending upon the cause.
- Give supplemental oxygen
- Noninvasive Positive-Pressure Ventilation (NPPV) can also be given.
- Intubation—put on the ventilator

Complications

- Prognosis is very poor.
- Mortality is between 40 to 60%.
- Pneumothorax development
- Permanent injury
- Pneumomediastinium
- Sepsis
- Renal failure
- Multiple organ failure

Cystic Fibrosis

A three year old Caucasian boy is brought in by his mom for what seems to be another episode on pneumonia. His mother recalls that he had trouble passing meconium when he was born.

Cystic Fibrosis (also known as Mucoviscoidosis) is an autosomal recessive disease due to a defect on the CFTR gene found on chromosome 7. More specifically, a frameshift mutation (3 base deletion) of the Phenylalanine at position 508. The CFTR gene normally codes for an anionic channel gated by ATP. When the ATP is hydrolyzed, cAMP is produced and is responsible for a cascade that activates proteins to regulate the channel. The CFTR channel normally secrets Chloride in certain organs such as the lungs and GI tract, but reabsorbs Chloride in other organs such as the skin. In cystic fibrosis, the CFTR protein is absent and the channel does not even exist on the membrane of the exocrine duct. Therefore, the cells (in the GI and lungs) can't absorb the Chloride and actually increase water and Sodium reabsorption, resulting in dehydrated thick mucous. This mucous is dehydrated to the point that it plugs organs and interferes with their normal function.

Looking at the case, it is important to note that cystic fibrosis is very common in Caucasians. In fact, it is the most common lethal genetic disease in whites. The manifestations of cystic fibrosis begin at birth. Most newborns will experience "meconium ilues," which is the inability of a newborn to pass meconium within the first 48 hours due to the obstruction of the distal ileum with this hard stool. Recurrent pulmonary infections such as pneumonia is common and is also due to the plugging of the lung parenchyma. This which creates a nice environment for the bacteria that will thrive in the absence mucocilliary clearance. Think about it, the little cilia are too weak to push the plugs up the airway for these patients to cough them out. CF patients are more susceptible to staph aureus and pseudomonas.

Other manifestations of the disease include mucous plugging in the pancrease resulting in a non functional organ (later in life). This leads to the regular manifestations of pancreatic insufficiency such as steatorrhea and deficiency of fat soluble vitamins. Another feature is failure to thrive due to recurrent infections and malabsorption. If these patients survive the infections and make it to adulthood, they can suffer of infertility due to bilateral absence of the vas deference in males and cilia of the fallopian tube in females. Sinus pain, nasal polyps, cough, hemoptysis, bronchiectasis, wheezing, intestinal obstruction and cirrhosis are also seen.

For a pregnant woman with a family history (or her husband's family history) of CF presents, the physician must recommend genetic screening early in the initial visit to make a diagnosis and lay out the options early in front of the family.

To diagnose CF, the physician must have strong clinical suspicion from birth. Meconium ileus in the newborn should prompt the physician to do workup and/or genetic testing. The gold standard for the diagnosis of CF is the Chloride sweat test (Chloride level above 60meq/L). It is important to note that the channel in the skin is normally doing the opposite of what it does in the internal organs such as the lungs. So instead of secreting Chloride (and then Sodium and water follows) into the lumen to keep the mucous moist, it normally absorbs Chloride (and then Sodium and water follow) from the skin to decrease water loss. In CF patients, since the channel is defective, not only will it fail to secrete Chloride into mucous secretions such as in the lung and pancrease (as we mentioned before), but it will also fail to absorb chloride from the skin, so Chloride, Sodium and water will remain in a high concentration on the skin, and the baby will taste salty. CXR and Chest CT will show bonchiectasis and scarring. The result of pulmonary function testing varies, but usually show a mixed pattern.

To manage CF patients, daily postural drainage and percussion to get as much mucous out as possible. This will help decrease the rate of infections. Bronchodilators are given as needed. Antibiotics are given during infections and/or for prophylaxis (usually with azithromycin). Inhaled rhDNase is a deoxyribonuclease to help break the mucous plugs. Another treatment to help loosen the mucous plugs is N-acetylcysteine which cleaves disulfide bonds. Finally, pneumococcal and influenza vaccinations must be given. The last resort is a lung transplant.

Title: Cor Pulmonale
Objectives for learning: Definition, Causes/ Risk factors, Clinical symptoms and signs, Diagnosis, Treatment

Definition:
It is basically right ventricular hypertrophy and right ventricular failure secondary to pulmonary hypertension due to any pulmonary disease. There is no heart issue involved.

Causes/ Risk factors:
- Chronic obstructive pulmonary disease (COPD)—the most common cause that leads to pulmonary hypertension. COPD patients usually die from cor pulmonale. They undergo left ventricular failure because of the chronic pulmonary hypertension.
- Recurrent PE
- Interstitial lung disease (ILD)
- Asthma
- Sleep apnea
- Pneumoconiosis

Pathophysiology
Pulmonary hypertension is the pressure inside the pulmonary artery. It is greater than 25 mmHg at rest and greater than 30 mmHg at exertion. With increase pressure inside the pulmonary artery, it leads to right ventricular hypertrophy and eventually failure due to pulmonary hypertension.

Clinical Symptoms And Signs
- Decrease exercise intolerance
- Cyanosis and digital clubbing
- Signs of Right ventricular heart failure—elevated jugular venous pressure, hepatomegaly, ascites and peripheral edema,
- Polycythemia

Diagnosis
- Chest x-ray: enlarged PA, RV, RA
- EKG: Right axis deviation, peaked p waves, and right ventricular hypertrophy.
- Echocardiogram: Right ventricular dilation is found. It is also used to rule out left ventricular dysfunction.

Treatment
Since COPD is the cause, so treat it. Give oxygen, bronchodilators, steroids, and ipratropium bromide. Use diuretics carefully in such patients. If a patient is suffering from right ventricular failure, give digoxin.

Title: Primary Pulmonary Hypertension

Objectives For Learning: Definition, Causes/ Risk factors, Pathophysiology, Clinical symptoms and signs, Diagnosis and Treatment.

Definition:

A mean BP greater than 25 mmHg at rest or less than 30 mHg during exertion is called pulmonary hypertension It is common in middle age woman. It is a diagnosis of exclusion. The survival is 2 to 3 years.

Causes/ Risk factors:

- Unknown

Pathophysiology

A thickening and the constriction develop within the arterial walls. The pressure inside the pulmonary artery increases, thus more thickening develops. Right ventricle works harder to push blood leading to right ventricular hypertrophy and eventually right ventricular failure.

Clinical symptoms and signs

- Dyspnea
- Fatigue
- Exertional chest pain
- Exertional syncope

Sign

- Loud P_2

These patients eventually develop right ventricular failure and it is manifested with the following symptoms and signs.

- JVD
- Ascites
- Hepatomegaly
- Peripheral lower extremity Edema

Diagnosis

- EKG: right axis deviation is present.
- Echocardiogram Dilated pulmonary artery, right atrium and the right ventricle may be present

- Pulmonary function tests: They show restrictive lung disease pattern with FEV/FVC may either be normal or high.

Treatment

Treatment is available for patients with primary pulmonary hypertension. Different medicines can be given such as

- **Pulmonary vasodilators:** IV prostacyclin (Epoprostenol)
- **Calcium channel blockers:** These decrease the pulmonary vascular resistance.
- Before performing a vasodilator trial, ask the patient to inhale Nitric oxide (NO), or give IV adenosine or oral calcium channel blockers.
- **Anticoagulant therapy:** Warfarin is also given to prevent thrombosis formation and keep their INR between 2 to 3.
- **Lung transplant:** This is the ultimate treatment of patients with primary pulmonary hypertension.

Title: Pulmonary Hypertension

Objectives For Learning: Definition, Causes/ Risk factors, Pathophysiology, Clinical symptoms and signs, Diagnosis, and Treatment

Definition:

A mean BP greater than than 25 mmHg at rest or less than 30 mHg when someone is exercising is called pulmonary hypertension.

Causes/ Risk factors:

Problem in pressure

- Mitral valve stenosis
- Atrial myxoma
- Left ventricular failure

Hyperkinetic

- Ventricular septal defect
- Atrial septal defect
- Left to right shunts
- Patent ductus arteriosus

Obstruction

- Pulmonary embolism
- Pulmonary artery stenosis

Obliteration

- Primary pulmonary hypertension
- CREST syndrome

Vasoconstrictive

- COPD
- Obstructive sleep apnea

Pathophysiology

The normal BP inside the pulmonary artery is less than 25mmHg. Pulmonary hypertension develops when something happens within the left atrium. It may include mitral valve stenosis, causing pressure inside the left atrium goes up and an eventually pulmonary pressure rises. Atrial myxoma is also responsible. Left ventricular failure also leads to the development of pulmonary hypertension. Other causes are mentioned above.

Clinical Symptoms And Signs

- Shortness of breath
- Fatigue
- Exertional syncope
- Exertional chest pain

Signs

- Loud P2
- Low A2

These patients eventually develop right ventricular failure and it is manifested with the following symptoms and signs.

- JVD
- Ascites
- Hepatomegaly
- Peripheral lower extremity Edema

Diagnosis

EKG: It may show right ventricular hypertrophy or right axis deviation.
Echocardiogram: Dilated pulmonary artery, right atrium and the right ventricle may be present.
Right heart catherization: It may show increased pulmonary pressure.

Treatment

Treat the underlying cause.

- If VSD is the cause, close up the septum.
- If AVD is the cause, fix it,
- In case of mitral valve, replace it.
- If right ventricular failure, it needs treatment.

Title: Goodpastures Syndrome

Objectives for learning: Definition, Cause, Clinical symptoms, Diagnosis and Treatment.

Definition:

An autoimmune antibodies IgG against alveolar basement membrane is the goodpasture syndrome. These antibodies attack and bound to the glomerular basement membrane. Type II hypersentivity reaction takes place.

Causes/ Risk factors:

- Autoimmune antibodies IgG

Pathophysiology

Clinical symptoms and signs

- Hemoptysis
- Dyspnea
- Cough
- Fever

Later on patients develop rapid progressive glomerulunephritis which leads to renal failure.

Diagnosis

Renal biopsy is done to look for the antibodies and glomerular basement membrane.

Treatment

The prognosis is very poor.

1) It is however treated by corticosteroids to decrease inflammation.
2) Plasmapheresis is done to take out the antibodies from the body.
3) Cyclophosphamide is also administered. Its side effect is the hemorrhagic cystitis. Melasma is the drug of choice in case patient develops hemorrhagic cystitis.

Title: Interstitial Lung Disease (ILD)- Introduction

Objectives For Learning: Definition, Pathophysiology, Types, Clinical symptoms and signs, and Diagnosis

Definition:

The space between the alveoli and the pulmonary capillaries is called interstitium. Anything that disturbs the ability of diffusion of oxygen and carbon dioxide across the membrane leads to diseases called ILD.

Causes/ Risk factors:

Pathophysiology

Chronic inflammation and fibrosis of the lungs parenchyma and the interstitium leads to interstitial lung disease. The fibrosis of the interstitium space and the parenchyma prevents the carbon dioxide to go out to the alveoli and diffusion of oxygen is also affected.

Types

- Idiopathic pulmonary fibrosis
- Sarcoidosis
- Pneumoconiosis
- Asbestosis
- Silicosis
- Drug induced (Bisophen, Bleomycin, Amiodarone, Neomycin and Gold)
- Radiation pneumonitis
- Cryptogenic organising pneumonia
- Wegener's granulomatosis
- Churg–Strauss syndrome
- Goodpasture's syndrome

Clinical Symptoms And Signs

- Shortness of breath on exertion, Later one dyspnea at rest
- Non productive cough
- Fatigue

On Physical Examination:

- Rales are heard on auscultation
- Digital clubbing
- Increased pulmonary vascular resistance leads to pulmonary hypertension and then cor pulmonale develops

Diagnosis

- Chest x-ray: it shows reticular, reticulo- nodular, ground glass appearance or honeycomb appearance.
- CT scan
- PFT = FEU_1/FVC = Very high – pointing towards development of restricted lung diseases
- DL_{CO} is very low.
- Tissue biopsy (via Fiberoptic bronchoscopy**)**

Treatment

Title: Pneumoconiosis

Objectives For Learning: Definition, Causes/ Risk factors, Pathophysiology, Clinical symptoms and signs, signs and symptoms of right ventricular failure, and Diagnosis.

Definition:

Accumulation of dust in the lung is called pneumoconiosis.

Causes/ Risk factors:

A cause or dust may be:

- Coal
- Beryllium
- Asbestos
- Silicon

Pathophysiology

The dust is taken up by the alveolar macrophages. They then cause inflammation and the interstitial fibrosis. The disease is a job related condition.

With the passage of time pulmonary vascular resistance develops, causing pulmonary hypertension and right ventricular failure.

Clinical Symptoms And Signs

- Dyspnea
- Productive cough
- Fatigue
- Exertional chest pain

On physical exam

- Digital clubbing

Signs and Symptoms of right ventricular failure

- Jugular venous distention (JVD)
- Ascites
- Hepatomegaly
- Peripheral lower extremity Edema

Diagnosis

- Pulmonary functions tests: There is a decreased FEV to FVC but the ratio is high. There is also a decreased diffusion capacity because of the fibrotic interstitium.
- Chest x ray shows ground glass appearance.

Treatment

Title: Asbestosis

Objectives for learning: Definition, Pathophysiology, Clinical symptoms and signs, Diagnosis and Treatment.

Definition:

Accumulation of dust (asbestos) in the lungs is called asbestosis.

Causes/ Risk factors:

Pathophysiology

There is an inflammation and fibrosis of the lung parenchyma.

The most common cancer in patients exposed to asbestos is bronchogenic carcinoma. Smoking increases the risk of development of bronchogenic carcinoma. The patients with asbestosis can also develop mesothelioma.

Clinical symptoms and signs

- Shortness of breath with exertion
- Productive cough
- Chest pain
- Eventually Respiratory failure develops

Diagnosis

- On chest x-ray, pleural thickening is present. It may be diffused or localized.
- There is a decreased diffusion capacity of carbon monoxide and an increased A-aC due to fibrosis.
- On biopsy ferruginous bodies are seen.

Treatment

Supportive treatment—ask patients to stop smoking

Title: Berryliosis

Objectives For Learning: Definition, Pathophysiology, Diagnosis and Treatment

Definition:

Patients exposed to beryllium develop berryliosis.

Causes/ Risk factors:

Pathophysiology

Berryliosis occurs in acute and in chronic form. The acute form may cause pneumonitis (an inflammation of airways). In chronic form one may find presence of granuloma, skin lesion, and hypercalcemia.

Clinical symptoms and signs

Diagnosis

Beryllium lymphocyte proliferation test

Treatment

Corticosteroid is the treatment of choice for both acute and chronic form of berryliosis.

Title: Coal Worker Disease (CWP)

Objectives for learning: Definition, Pathophysiology, Complications and Clinical symptoms and signs

Definition:

It is also called black lung disease. Accumulation of coal within the lungs leads to a condition known as coal worker disease (CWP).

Causes/ Risk factors:

Pathophysiology

The alveolar macrophages engulf the coal particles, cause chronic inflammation and eventually develop fibrosis. Coal is however least fibrogenic than silicon, asbestos or beryllium. The alveolar macrophages develop central lobular emphysema usually in the respiratory bronchiole.

Complications

- Fibrotic opacities inside the lung--- a crippling disease
- Cor pulmonale
- Caplan syndrome

Clinical symptoms and signs

- It may be asymptomatic or symptomatic.

Diagnosis

Treatment

Title: Silicosis

Objectives for learning: Definition, Causes/ Risk factors, Diagnosis, Complications, and Treatment

Definition: accumulation of silicon (sand) within the lung leads to a condition known as silicosis. Silicon is highly fibrogenic.

Causes/ Risk factors:

People who work in:

- Foundries
- Sand blasting
- Mines

Pathophysiology

The patients constantly inhaled sand, which is picked up by the alveolar macrophages which causes inflammation and fibrosis of the lungs.

Clinical symptoms and signs

Diagnosis

Chest x-ray: Silicones are present in nodualar form or egg shell calcifications in the hilar lymph nodes are seen.

Complications

- Increased risk of developing lung cancer
- Tuberculosis
- Caplan syndrome (it is the pneumoconiosis (silicosis) plus large rheumatoid nodule inside the lung)

Treatment

- Patients usually die of progression of the pulmonary diseases.
- Yearly ppd (purified protein test) is necessary to be done yearly in such patients. If ppd is greater than 10mm, isoniazid is started as a prophylactic measure. Pyridoxine is also given along with isoniazid so to prevent the development of peripheral neuropathy.

Title: Lung Cancer

Objectives for learning: Types, Causes/ Risk factors, Staging of Non small cell lung cancer (NSCLC), Clinical Symptoms and Signs, Local Invasion of the Tissues, Metastasis, Paraneoplastic Syndrome, Diagnosis and Treatment

Definition:

Types:

There are two types of lung cancer:

1. Small cell lung cancer (SCLC)

25% of total lung cancers are the small cell cancer. They are centrally located tumors.

2. Non small cell lung cancer (NSCLC)

75% of total lung cancers are the non small cell cancer. These include:

- Squamous cell
- Adenocarcinoma
- Large cell lung carcinoma
- Bronchioalveolar cell lung carcinoma

Causes/ Risk factors:

- Smoking (85% cases develops lung cancer due to smoking)
- Passive smoking
- Asbestos exposure (Bronchogenic carcinoma and mesothelioma may occur with asbestos exposure)

Staging

T = size of the tumor

N= Lymphatic infiltration

M= Metastasis

Staging of Non small cell lung cancer (NSCLC)

Twp approaches are used:

- Limited (Chest and supraclavicular lymph nodes)
- Extended (outside the chest and the supraclavicular lymph nodes)

Pathophysiology

Clinical Symptoms And Signs

Non specific symptoms are usually present. Other includes:

- Airway involvement—cough, hemoptysis, wheezing, shortness of breath.
- Recurrent pneumonia
- Weight loss
- Anorexia

Local Invasion Of The Tissues

- Superior Vena Cava Syndrome – presents with facial swelling ,arm swelling, distention of juglar vein
- Phrenic Nerve Palsy – presents with hemidysphagia
- Recurrent Laryngeal Nerve Palsy
- Horner Syndrome—presents with miosis, anhydrosis and ptosis
- Pancoat Tumor—shoulder arm pain. Squamous cell carcinoma is also very common here.

Metastasis

- Bone
- Brain
- Liver
- Abdomen

Paraneoplastic Syndrome

Small cell lung carcinoma is associated with paraneoplastic syndrome.
It can produce:

1. Anti-diuretic hormone which can causes development of syndrome of inappropriate syndrome.
2. Ectopic Adrenocorticotropic hormone
3. Eaton lambert syndrome --presents with proximal muscle weakness fatigue and loss of deep tendon reflexes.
4. Hypertrophic pulmonary osteoarthropathy: It is more common in adenocarcinoma and squamous cell lung carcinoma. This syndrome causes severe bone pain, usually long bone pain. Digital clubbing also occurs here.

Squamous cell carcinoma

- It produces Parathyroid like related peptide hormone (PTH r P)

Diagnosis

- Chest x ray: It is most important here.

- CT scan: It is very useful for staging of the cancer. It helps us to see lymphoadenopathy. It enables detection of local or distant metastasis.
- Cytological examination of sputum
- Fiberoptic bronchoscopy
- Transthoracic needle biopsy
- Mediastinoscopy

Treatment

Small Cell Lung Carcinoma Treatment

- ✓ They are so small that cannot be surgically resected. Chemotherapy is the treatment of choice. Radiotherapy can also be given.

Non Small Cell Lung Carcinoma Treatment

- ✓ Surgery is important here.
- ✓ Radiation therapy is needed following surgery.
- ✓ Chemotherapy can be given.

Prognosis

The prognosis is very poor. The 5 year survival of patients with lung cancer is only 14%. 85% patients with small cell lung cancer present with an extensive disease usually die within 2 years of presentation.

Title: Pleural Effusions

Objectives For Learning: Definition, Causes/ Risk factors, Types of pleural effusion, Light's Criteria Exudative Effusions, Clinical Symptoms and Signs, Diagnosis, Types Of Pleural Fluids, Parapneumonic Effusion VS Empyema and Treatment.

Definition:

Excessive accumulation of fluid in pleural space is called pleural effusion.

Causes/ Risk factors:

- Increased drainage
- Increased production of fluid by pleural space cells
- Decrease drainage
- Congestive heart failure is by far the most common cause of pleural effusion particularly trasudative.
- Pneumonia is another common cause.

Pathophysiology

Types of Pleural Effusion

Transudation: (watery fluid): It is because of increased hydrostatic pressure and decreased osmotic pressure (due to protein loss). Transudation occurs in diseases such as:

- Chronic renal failure
- Nephrotic syndrome
- Cirrhosis
- Hypoalbuminemia
- Peritoneal dialysis
- Pulmonary embolism

Exudation: (pus, hemorrhagic, milky): It occurs in:

- Bacterial infection/ pneumonia causing parapneumonic effusion
- TB
- Malignancy / Metastasis
- Pulmonary embolism
- Collagen vascular diseases

Light's Criteria Exudative Effusions

	Transudative	Exudatative
LDH effusion	<200Iu/ml	>200 Iu/ml
LDH effusion / LDH Serum	<0.6	>0.6

Protein effusion/ protein serum	<0.5	>0.5

LDH (lactate dehydrogenase)

Clinical Symptoms And Signs

It may be asymptomatic.
A person may experience:

- Dyspnea on exertion
- Peripheral edema
- Paroxysmal nocturnal dyspnea (PND)
- Orthopnea

On physical examination
There is:

- Dullness on percussion
- Decrease breath sounds
- Decrease tactile fremitus

Diagnosis

- Chest x-ray: There is blunting of costophrenic angle.
- CT scan of chest
- Thoracentesis
- CBC with differential
- Chemistry—glucose and proteins
- Cytology
- Cell count
- ph of the pleural fluid
- Amylase of the pleural fluid
- Gram stain of the pleural fluid
- LDH of the pleural fluid and protein
- PCR
- Pleural biopsy

  - ✓ Esophageal rupture can lead to high amylase level in pleural fluid; pancreatitis can also cause elevation of amylase.
  - ✓ If glucose level in pleural fluid is less than 60, always think about rheumatoid arthritis.
  - ✓ If lymphocytes are present in pleural fluid, they may indicate TB.

Types Of Pleural Fluids

- Milky effusion: A fluid may be milky due to chylothorax or lymphatic obstructions.
- Pus: empyema
- Hemorrhagic effusion: Trauma, malignancy

- Parapneumonic effusion: pneumonia

Parapneumonic Effusion VS Empyema

- **Parapneumonic Effusion:** This is a non-infected pleural fluid. It is treated by antibiotics. This is the uncomplicated effusion.
- **Empyema:** Empyema is an infected pleural effusion. Chest tube is also required here for drainage along with antibiotics. Thrombolytics can be injected via intrapleural injections. Empyema is the complicated pleural effusion. It can cause mediastinitis. Surgical lysis of the adhesion may sometimes be needed.

Treatment

Transudative Effusion

- ✓ Give diuretics (furosemide) and restrict salt intake. Therapeutic thoracocentesis can be done.

Exudative Effusion

- ✓ Treat the underlying cause.

Respiratory Physiology: Flow-Volume loops

Tidal Volume (TV): It is the volume inspired or expired with each normal breath.

Inspiratory Reserve Volume (IRV): Volume of air that can be inspired over and above tidal volume.

Expiratory Reserve Volume (ERV): It is the maximum volume of air that can be breath out from the end-expiratory position.

Residual Volume (RV): It is the volume that remains after maximum expiration. It is always inside the lungs.

Inspiratory Capacity (IC): It is the sum of tidal volume and inspiratory reserve volume.

IC= TV + IRV

Vital Capacity (VC): It is the maximum amount of air that can be actually blow out.

VC= IRV +TV+ERV

Functional Residual Capacity (FRC): It is the volume remaining in the lung after a tidal volume is expired.

FRC= RV+ERV

Total Lung Capacity (TLC): It is the sum of IRV, RV, TV and ERV.

TLC= IRV+RV+TV+ERV

Forced Expiratory Volume (FEV_1): It is the volume of air that can be expired in the first second of a forced maximal expiration.

Forced Vital Capacity (FVC): It is the sum of tidal volume expiratory reserve volume and inspiratory reserve volume.

FVC= TV+ ERV+IRV.

The ratio of FEV1 to FVC will tell the patient has whether the restrictive pattern or interstitial lung disease.

- In a normal person $FEV_1/FVC= 0.8= 80\%$ i.e. 80% of the forced vital capacity.
- In case of an Obstructive Disease $FEV_1/FVC= 6/10=60\%$

FEV_1 is reduced more than FVC.

- In case of a Restrictive Disease

Both FEV_1 and FVC are reduced while their ratio can be increased or can be normal. Functional residual volume (FRV) is also decreased in a restrictive lung disease.

Title: Pneumonia

Objectives for learning: Definition, Community acquired pneumonia, Nosocomical infection, Typical Community acquired pneumonia, and Atypical Community acquired pneumonia, Causes, Diagnosis and Treatment.

Definition:

There are two types of pneumonia:

1. Community acquired pneumonia
2. Nosocomcal pneumonia

Community acquired pneumonia is the pneumonia acquired from the community or outside the environment. Its second definition is any pneumonia which is developed within 72 hours of hospitalization.

Nosocomical infection is the infection acquired within 72 hours of hospitalization.
Vaccination decreases the risk of development of pneumonia. Pneumococcal vaccine is available. It is given to elder over age 65 years, younger patients or patients with sickle cell disease, heart diseases, pulmonary disease or to diabetics. It is also given to aspleenic individuals because these are susceptible to develop bacterial infections.

Community acquired pneumonia is of two types:

1) Typical Community acquired pneumonia
2) Atypical Community acquired pneumonia

Typical Community acquired pneumonia

The most common typical pneumonia is the streptococcus pneumonia.
Organisms involved are:

- Streptococcus
- Haemophilus influenzae
- Aerobic gram negative rods
- Klebsiella
- Enterobacteriaceae
- Staphylococcus aureus

Clinical symptoms and signs

Typical Community acquired pneumonia is presented with acute onset of fever and shaking chills. They also complain of productive cough. Sputum is thick and purulent. Pleuritic chest pain suggests a pleural effusion is also present.

On examination: Tachycardia is present, they are tachypnic and late respiratory crackles are heard. Also, they have bronchial breath. Increased tactile fremitus plus dullness to percussion is present. Pleural friction rib is also found when they have pleural fluid. It is diagnosed by the help of Chest x-ray, which may show lobar consolidation. If more lobes are involved then it is known as multilobar pneumonia, which is a serious disease.

Atypical Community Acquired Pneumonia

It is presented with insidious or acute onset.

Clinical symptoms and signs

- Headache
- Sore throat
- Fatigue
- Myalgia
- Dry cough
- Fever

Signs:

- Pulse - temperature dissociation
- Normal pulse
- Wheeze
- Ronchi
- Crackles

Diagnosis

Chest x ray--- diffuse reticulonodular appearance infiltrates are seen.
Absent or minimal consolidations

Causes

Organisms involved here are:

Bacteria:

- Mycoplasma pneumonia
- Chlamydia pneumonia
- Chlamydia psittaei

- Coxiella burneti (Q fever causing organism)
- Legionella

Viruses:

- Parainfluenza
- Adenovirus
- Influenza
- Respiratory syncytial virus

Diagnosis of Pneumonia

- PA/ lateral view chest X-ray
- Sputum culture
    - Good sputum: >25 PMN's, < 10 epithelial cells
- Gram stain
- CBC
- Basic metabolic profile
- BUN/Creatinine
- Glucose
- Oxygen saturation
- Blood culture
- Antibiotics

Important Notes

1. Alcoholics: Klebsiella
2. Immigrants: TB
3. Nursing home (Female, 50 years): Nosocomial infection
    - Pseudomonas
        - Pneumonia
        - Sepsis
        - Diabetic osteomyelitis
        - Urosepsis
        - Sinusitis
4. HIV: Pneumocystis carinii, mycobacterium tuberculosis
5. Organ transplant: Legionella
6. Renal failure or smokers: Legionella

Special Stains

- For TB, acid fast stain is used
- For pneumocystis carinii, silver stain is used.
- For Legionella, urinary antigen assay is used.

Treatment

1. Outpatient therapy includes:
   - Macrolides are good antibiotics such as doxycycline or azithromycin
   - Fluroquinolones such as levofloxacin are also good for them.

For patients with above age 60 years, 2nd and 3rd generation cephalosporins are useful.

   - Amoxicillin/clavulanic acid or Fluroquinolones such as levofloxacin are also good.
2. Inpatient therapy includes levofloxacin plus 3rd generation cephalosporin

Complications of Pneumonia

- Pleural effusions
- Empyema
- Respiratory failure

Tuberculosis

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Let us start with some basic microbiology. Mycobacterium tuberculosis (tb) is a non motile organism, with no capsule and no pilli. It stains red with acid fast stain, as well as the Auramine rhodamine stain. It is an obligate aerobe, characterized by slow growth. The important virulence factors are:

a) Mycolic acid- large fatty acids in the cell membrane make it resistant to drying.

b) Cord factor- helps block neutrophil migration and allows growth in serpentine pattern. This is the most important virulence factor because strands missing it will not cause disease.

c) Sulfatides- blocks phagosome/lysosome fusion, allowing tb to be facultative intracellular.

Mycobacterium tb is cultured in Lowenstein Jensen medium (slow growth).

Primary tb occurs when the organism in inhaled (problem in airplanes where air circulates) into the lower part of the upper lobe of the upper part of the lower lobe. Possible outcomes after primary exposure depend on the immune status of the patient. In immunocompromised, tb symptoms begin right away. A healthy may able to completely eliminate the organism. A large group of people will not be able to eliminate it completely, but will be able to contain it via granuloma formation. Basically, cell mediated immunity is ON, T cells activate lots of macrophages and neutrophils, and a caseating granuloma (central necrosis) keeps tb locked. This is a very expensive process. T cells need lots of energy to "pay" the macrophages to form the granuloma. Usually, this is assymptomatic. It can be detected on an xray as Gohn complex (the calcified granuloma and the adjacent lymph node).

At any point, if the immune system gets weakend (ex. Chemo, old age….) T cells can no longer support macrophages and tb escapes to areas of high oxygen tension (upper lobes of lungs). This is reactivation of tb, and patients will now complain of weight loss, fever, night sweats and maybe hemoptysis. If not treated, tb can become "military tb," where it spreads to the brain, bone, liver and kidneys.

Diagnosis- start with PPD test to screen (ex. Annually in health care workers). If ever postive (>15mm in healthy, >5 in immunocompromised such as HIV+, >10 in those at risk such as health care workers), then order a chest xray, as well as sputum stain and culture. If CXR or sputum is positive, then treat this active tb. If they are negative but you have high clinical suspicion (from the positive PPD), then treat as latent tb. Just remember, false positive PPD is seen with patients who had BCG vaccine. False negative PPD can be seen in severely immunocompromised patients. Therefore, one must maintain high clinical suspicion.

Active tb treatment- treat for 6months total, the 4 drugs for 2 months then rifampin and isoniazid for 4 months. Extend the treatment to total of 9 months if military tb.

Latent tb treatment- isoniazid for 9 months.

Quickly, when treating, remember-

INH is always given with pyridoxine (B6) to prevent peripheral neuropathy.

Warn patients the rifampin causes red coloration of body secretions, this is nothing to be alarmed about.

Ethambutol can cause optic neuritis, so give lower dose if renal function is impaired.

Finally, give steroids to decrease risk of constrictive pericarditis.