Pathology of Pneumonia

Dr. Atif Ali Bashir
Assistant Professor of Pathology
College of Medicine
Majma'ah University

Introduction:

5000 sq meters of area….!
(olympic track)
Filters >10,000 L of air / day…!
Normal lungs are sterile.
Delicate, thin **resp. mem** – gas exch.
Filter, humidify, sterilize, highly **sensitive**.

RTI – Resp. tract inf. commonest in medical practice.
Enormous morbidity & mortality.
Pneumonia – inflammation of alveoli.

Normal Lung

Normal Lung

Etiology:

Decreased resistance - General/immune
Virulent infection - Lobar pneumonia
Defective Clearing mechanism
- Cough/gag Reflex – Coma, paralysis, sick.

Mucosal Injury – smoking, toxin aspiration
Low Alveolar defense - Immunodeficiency
Pulmonary edema – Cardiac failure, embol.
Obstructions – foreign body, tumors

Patterns of Lung disorders:

Airway

Bronchitis, Bronchiectasis, Bronchiolitis.
Tumors / Cancer

Parenchyma

Pneumonia.
Lung abscess, TB
Hyaline membrane dis (HMD & ARDS)

Pneumoconiosis
Tumors / Cancer

Pleura:

Pleural effusion (TB)
Tumors / Cancer

Pathogenesis of Pulmonary Infections

Step 1: Entry

Aspiration (ie Pneumococcus)

Inhalation (ie Mtb and viral pathogens)

Inoculation (contaminated equipment)

Colonization (in patients with COPD)

Hematogenous spread (patients with sepsis)

Direct spread (adjacent abscess)

Pathogenesis:

Pathogenesis:

Pneumonia Types:

Etiologic Types:

Infective

- Viral
- Bacterial
- Fungal

Tuberculosis

Non Infective

Toxins

chemical

Aspiration

Morphologic types:

Lobar

Broncho

Interstitial

Duration:

Acute

Chronic

Clinical:

Primary / secondary. Typical / Atypical Community a / hospital a

Lobar Pneumonia:

whole lobe, exudation - consolidation
95% - Strep pneum.(Klebsiella in aged, DM, alcoholics)
High fever, rusty sputum, Pleuritic chest pain.
Four stages: (*also in bronchopneumonia)

Congestion – 1d – vasodilatation congestion.
Red Hepatization 2d Exudation+RBC
Gray Hepatizaiton 4d neutro & Macrophages.
Resolution – 8d few macrophages, normal.

Pathogenesis of Pneumonia

Lobar Pneumonia:

Lobar Pneumonia:

Lobar Pneumonia – Gray hep…

Lobar Pneumonia:

Lobar Pneumonia: Congestion

Lobar Pneumonia:
Red hepat.
Lobar Pneumonia:
Grey hepat.
Broncho-pneumonia
Bronchopneumonia
(patchy)
Extremes of age.
(infancy and old age)
Staph, Strep, Pneumo
& H. influenza

Patchy consolidation – not limited to lobes.
Suppurative inflammation
Usually bilateral
Lower lobes common
Broncho-pneumonia
Broncho-pneumonia
Bronchopneumonia:
Bronchopneumonia - CT
Bronchopneumonia

Broncho – **Pneumonia**
- Lobar
Extremes of age.
Secondary.
Both genders.
Staph, Strep, H.infl.
Patchy consolidation
Around Small airway
Not limited by
anatomic boundaries.
Usually bilateral.
Middle age – 20-50

Primary in a healthy males common.
95% pneumoc (Klebs.)
Entire lobe consolidation
Diffuse
Limited by anatomic boundaries.
Usually unilateral
Broncho – **Pneumonia**
- Lobar

Interstitial / atypical Pneumonia

Primary atypical pneumonia in the immunocompetant host (Mycoplasma or Chlamydia)

Interstitial pneumonitis

immunocompromised host :
Pneumocystic carinii; CMV
Immunocompetant host: Influenza A

Gross features:

Lungs are heavy but not firmly consolidated

Microscopic features:

Septal mononuclear infiltrate
Alveolar air spaces either 'empty' or filled with proteinaceous fluid with few or no inflammatory cells

Interstitial Pneumonia:
Chronic Pneumonia
Chronic, lymphoid infiltrate,
No classic stages.
Lung destruction – cavity, abscess etc.
Organisms
- Mycobacterium tuberculosis

Histoplasma capsulatum
Aspergillosis
Actinomyces

Comm – **Pneumonia**

- Nosoc

In healthy adults

Gram positive.

Streptococcus pneumoniae (90%)

Strep. Pyogenes, Staph, H. influenzae

and Klebsiella in elderly or with COPD. In *sick patients. gram-negative bacilli Pseudomonas aeruginosa, Escherichia coli, Enterobacter, Proteus, and Klebsiella.

Pathogenesis of Clinical features:

*Alveolar inflammation. Tachypnoea, Dyspnoea, Resp Acidosis → Solid/airless lungs – decreased oxygenation.

Dull percussion - Consolidation – Exudation

Rusty sputum - RBC & Inflammatory cells.

Fever – Inflammatory mediators.

Complications of Pneumonia

Abscesses

Localized suppurative

necrosis, Right side often in aspiration. Staphylococcus; Klebsiella; Pneudomonas

Pleuritis / Pleural effusion.

Inflammation of the pleura (Streptococcus pneumoniae)

Blood rich exudate (esp. rickettsial diseases)

Empyema

Pus in the pleural space.

Septicemia

Abscess formation

Lung Abscess:

Abscess formation

Lung Abscess:

Lung Fungal Abscess: Candida