PNEUMONIA

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OUTLINE

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- Pathology
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- Treatment
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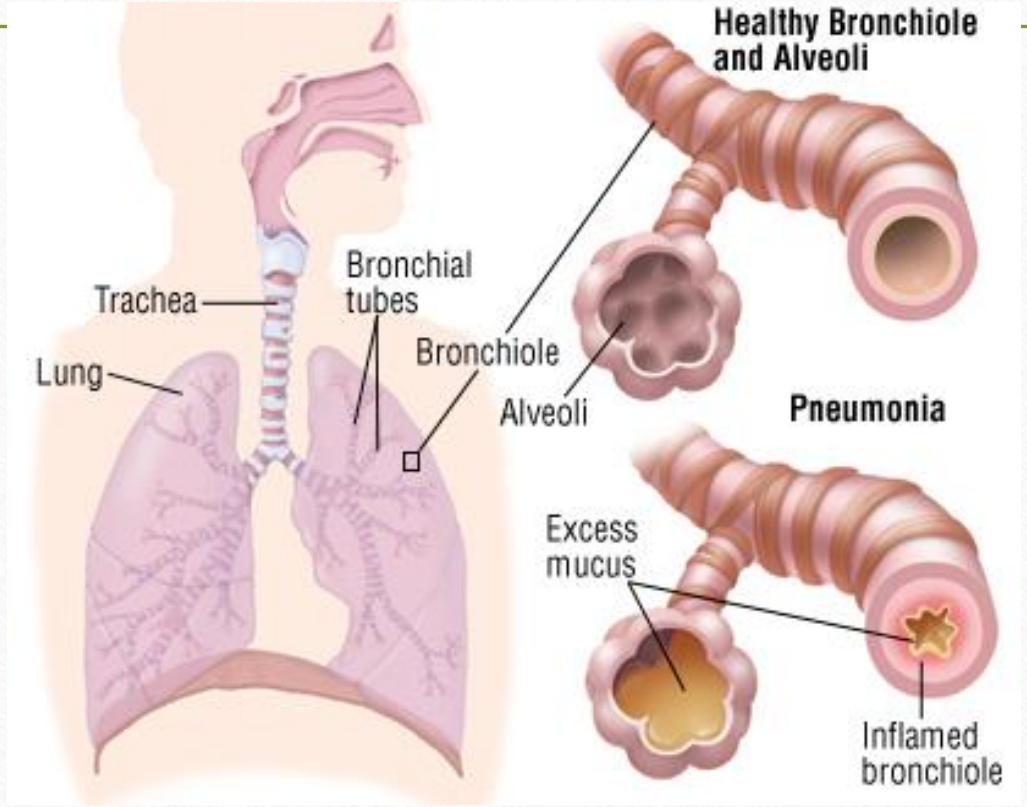
- Prognosis
- Prevention

INTRODUCTION

- Pneumonia is an infection of the pulmonary parenchyma



CLASSIFICATION

In the past, pneumonia was typically classified as:

1. Community-acquired (CAP)
2. Hospital-acquired (HAP)
3. Ventilator-associated (VAP)

CLASSIFICATION

- Over the past two decades, however, some persons presenting with onset of pneumonia as outpatients have been found to be infected with the multidrug-resistant (MDR) pathogens previously associated with HAP.
- Potential involvement of these MDR pathogens has led to a designation for a new category of pneumonia—*health care–associated pneumonia* (HCAP)

PATHOPHYSIOLOGY

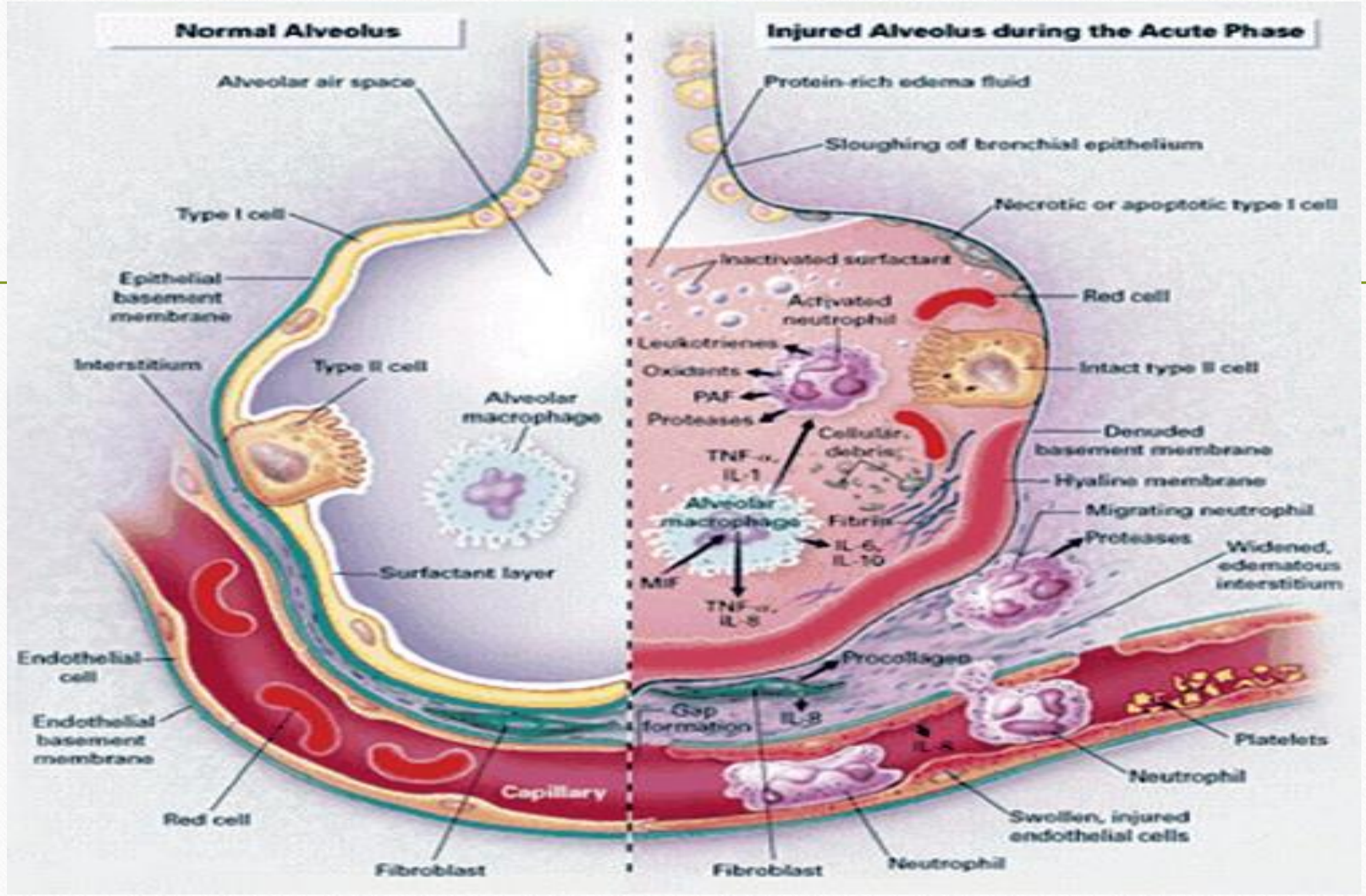
- Pneumonia results from
  - Proliferation of microbial pathogens at the alveolar level
  - Host's response to those pathogens
- Routes of infection:
  1) Aspiration from the oropharynx
  2) Inhalation of contaminated droplets
  3) Hematogenous spread (e.g., from tricuspid endocarditis)
  4) Contiguous extension from an infected pleural or mediastinal space

PATHOPHYSIOLOGY

- Host defence factors:
  - Mechanical: hairs and turbinates of the nares, branching architecture of the tracheobronchial tree
  - Mucociliary clearance
  - Local antibacterial factors
  - Gag reflex, cough reflex
  - Normal flora
  - Alveolar macrophages
- Only when the capacity of the alveolar macrophages to ingest or kill the microorganisms is exceeded does clinical pneumonia become manifest.

PATHOPHYSIOLOGY

- The host inflammatory response, rather than proliferation of microorganisms, triggers the clinical syndrome of pneumonia
- Alveolar macrophages initiate the inflammatory response
- Interleukin 1 and TNF release, results in fever
- Interleukin 8 and G-CSF, stimulate the release of neutrophils, producing both peripheral leukocytosis and increased purulent secretions.
- Inflammatory mediators released by macrophages and the newly recruited neutrophils create an alveolar capillary leak → radiographic infiltrate and rales detectable on auscultation, and hypoxemia results from alveolar filling.
- When even erythrocytes can cross the alveolar-capillary membrane, hemoptysis occurs


Normal Alveolus
Alveolar air space
Type I cell
Epithelial basement membrane
Interstitium
Type II cell
Alveolar macrophage
Surfactant layer
Endothelial cell
Endothelial basement membrane
Red cell
Capillary
Fibroblast
Injured Alveolus during the Acute Phase
Protein-rich edema fluid
Sloughing of bronchial epithelium
Necrotic or apoptotic type I cell
Inactivated surfactant
Activated neutrophil
Red cell
Leukotrienes
Oxidants
PAF
Proteases
Intact type II cell
Cellular debris
Denuded basement membrane
TNF, IL-1
Hyaline membrane
Alveolar macrophage
Fibrin
Migrating neutrophil
Proteases
IL-6, IL-10
MIF
TNF, IL-8
Widened, edematous interstitium
Procollagen
Gap formation
IL-8
Platelets
Neutrophil
Swollen, injured endothelial cells
Fibroblast
Neutrophil

PATHOPHYSIOLOGY

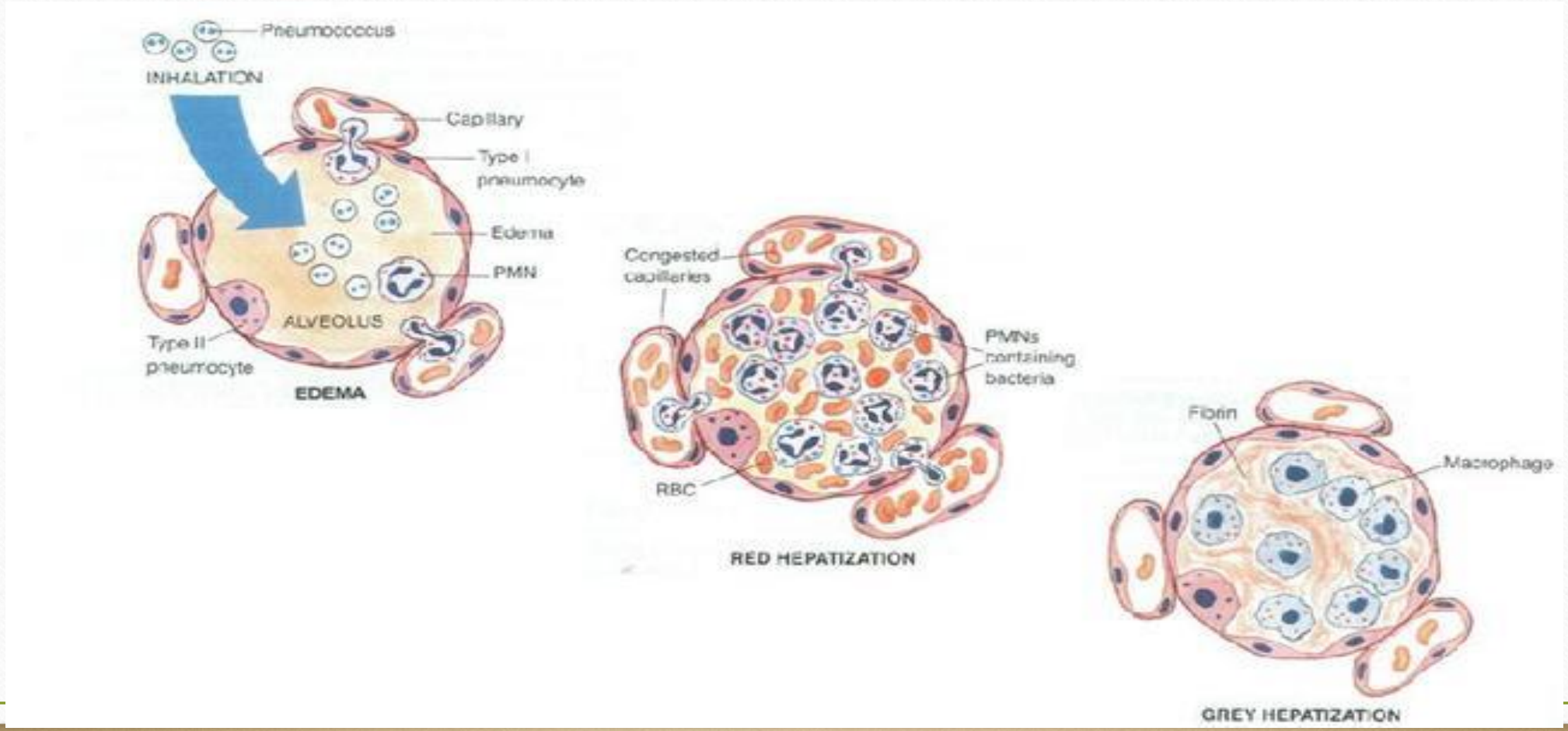
- Moreover, some bacterial pathogens appear to interfere with the hypoxemic vasoconstriction that would normally occur with fluid filled alveoli, and this interference can result in severe hypoxemia.
- Increased respiratory drive in the systemic inflammatory response syndrome leads to respiratory alkalosis.
- Decreased compliance due to capillary leak, hypoxemia, increased respiratory drive, increased secretions, and occasionally infection-related bronchospasm all lead to dyspnoea.
- If severe enough, the changes in lung mechanics secondary to reductions in lung volume and compliance and the intrapulmonary shunting of blood may cause respiratory failure and the patient's death.

PATHOLOGY

Lobar pneumonia typically follows four stages:

Stage	Macroscopic appearance	Microscopic appearance
Congestion (first 24 hours)	The affected lobe is red, heavy and boggy	Vascular dilatation Alveolar exudate contains mostly bacteria
Red hepatization (days 2-3)	Red, firm lobe (liver-like consistency)	Alveolar exudate contains erythrocytes, neutrophils, and fibrin
Gray hepatization (days 4-6)	Gray-brown firm lobe	RBCs disintegrate Alveolar exudate contains neutrophils and fibrin
Resolution	Restoration of normal architecture	Enzymatic digestion of the exudate

PATHOLOGY


Pneumococcus
INHALATION
Capillary
Type I pneumocyte
Edema
PMN
ALVEOLUS
Type II pneumocyte
EDEMA
Congested capillaries
PMNs containing bacteria
RBC
RED HEPATIZATION
Fibrin
Macrophage
GREY HEPATIZATION

ETIOLOGY

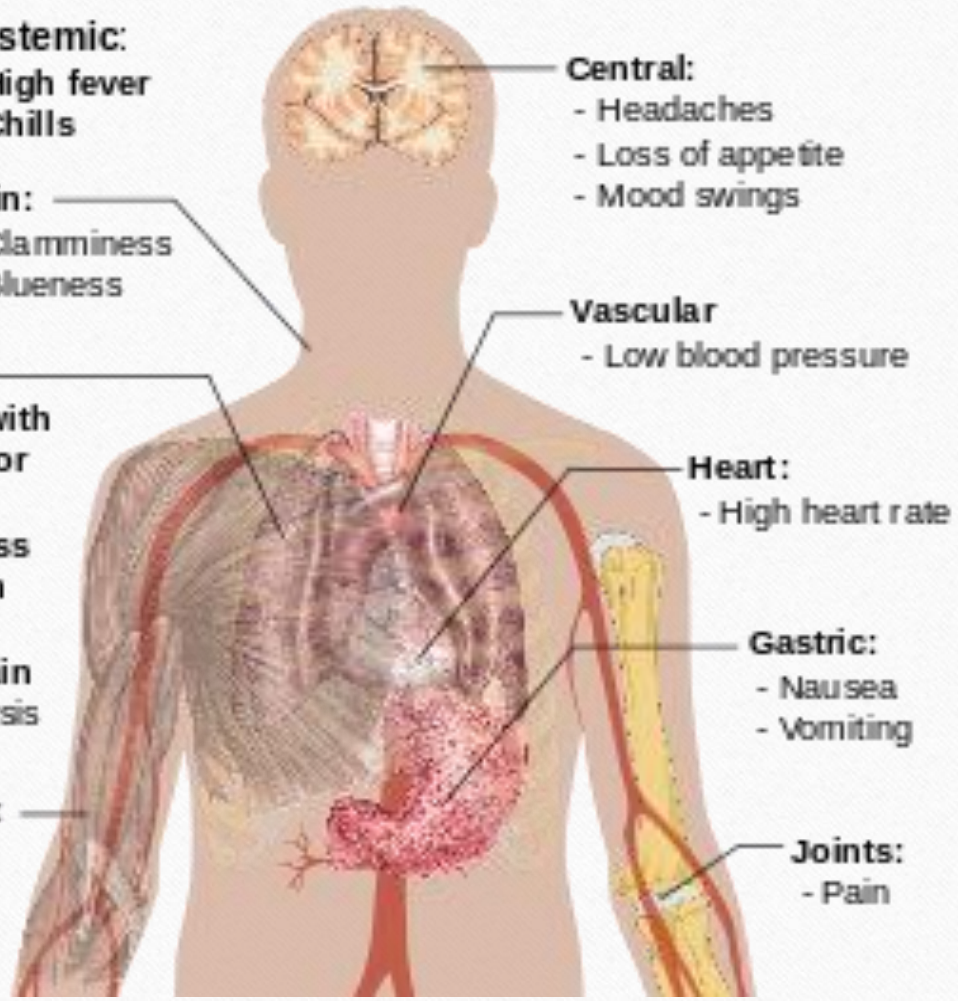
Outpatients	Non ICU	ICU
Streptococcus pneumoniae	S. pneumoniae	S. pneumoniae
Mycoplasma pneumoniae	M.pneumoniae	Staphylococcus aureus
Haemophilus influenzae	Chlamydia pneumonia	Legionella spp
C. pneumoniae	H. influenzae	Gram-negative bacilli
Respiratory viruses	Legionella spp	H. influenzae
	Respiratory viruses	

Epidemiologic Factors Suggesting Possible Causes of CAP

Factor	Possible Pathogen(s)
Alcoholism	Streptococcus pneumoniae, oral anaerobes, Klebsiella pneumoniae, Acinetobacter spp., Mycobacterium tuberculosis
COPD and/or smoking	Haemophilus influenzae, Pseudomonas aeruginosa, Legionella spp., S. pneumoniae, Moraxella catarrhalis, Chlamydia pneumoniae
Structural lung disease (e.g., bronchiectasis)	P. aeruginosa, Burkholderia cepacia, Staphylococcus aureus
Dementia, stroke, decreased level of consciousness	Oral anaerobes, gram-negative enteric bacteria
Lung abscess	CA-MRSA, oral anaerobes, endemic fungi, M. tuberculosis, atypical mycobacteria

Main symptoms of infectious

Pneumonia



CLINICAL MANIFESTATIONS

- Vary from indolent to fulminant in presentation and from mild to fatal in severity

Symptoms:

- History of fever with chills and/or sweats
- Cough may be either non-productive or productive of mucoid, purulent, or blood-tinged sputum
- Gross hemoptysis is suggestive of CA-MRSA pneumonia
- If the pleura is involved, the patient may experience pleuritic chest pain
- 20% of patients may have GI symptoms such as nausea, vomiting, and/or diarrhea
- Fatigue, headache, myalgias, and arthralgias
- Elderly: new onset confusion

CLINICAL MANIFESTATIONS

Signs:

- Febrile
- Tachycardia
- Increased respiratory rate
- Use of accessory muscles of respiration
- Tactile fremitus: increased or decreased reflecting underlying consolidated lung and pleural fluid, respectively
- Percussion note can vary from dull to flat, reflecting underlying consolidated lung and pleural fluid, respectively.
- Auscultation: Crackles, bronchial breath sounds, and possibly a pleural friction rub
- Severely ill patients may have septic shock and evidence of organ failure

DIFFERENTIAL DIAGNOSIS

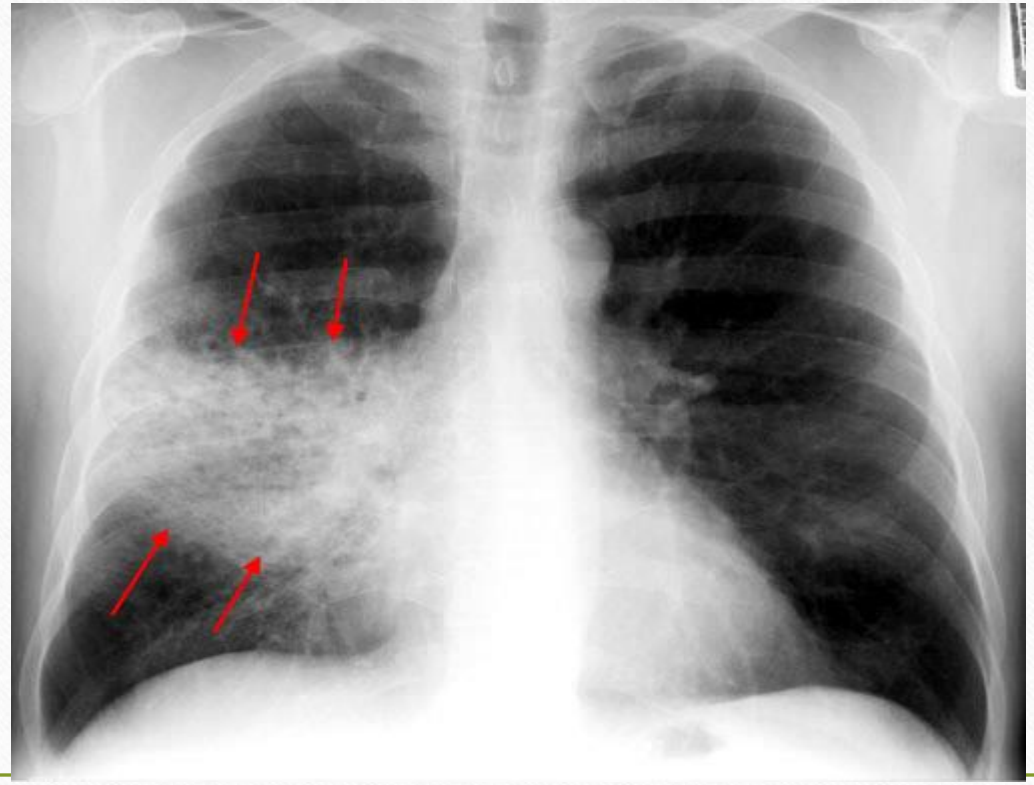
- Acute bronchitis
- Acute exacerbations of chronic bronchitis
- Heart failure
- Pulmonary embolism
- Hypersensitivity pneumonitis
- Radiation pneumonitis

DIAGNOSIS

- History and examination
- Chest radiograph: pneumatoceles suggest infection with *S. aureus,* and an upper-lobe cavitating lesion suggests tuberculosis
- CT scan: suspected postobstructive pneumonia caused by a tumor or foreign body or suspected cavitary disease
- Sputum Gram's stain and culture: yield of positive cultures from sputum samples is ≤50%
- Deep-suction aspirate or bronchoalveolar lavage sample in intubated patients

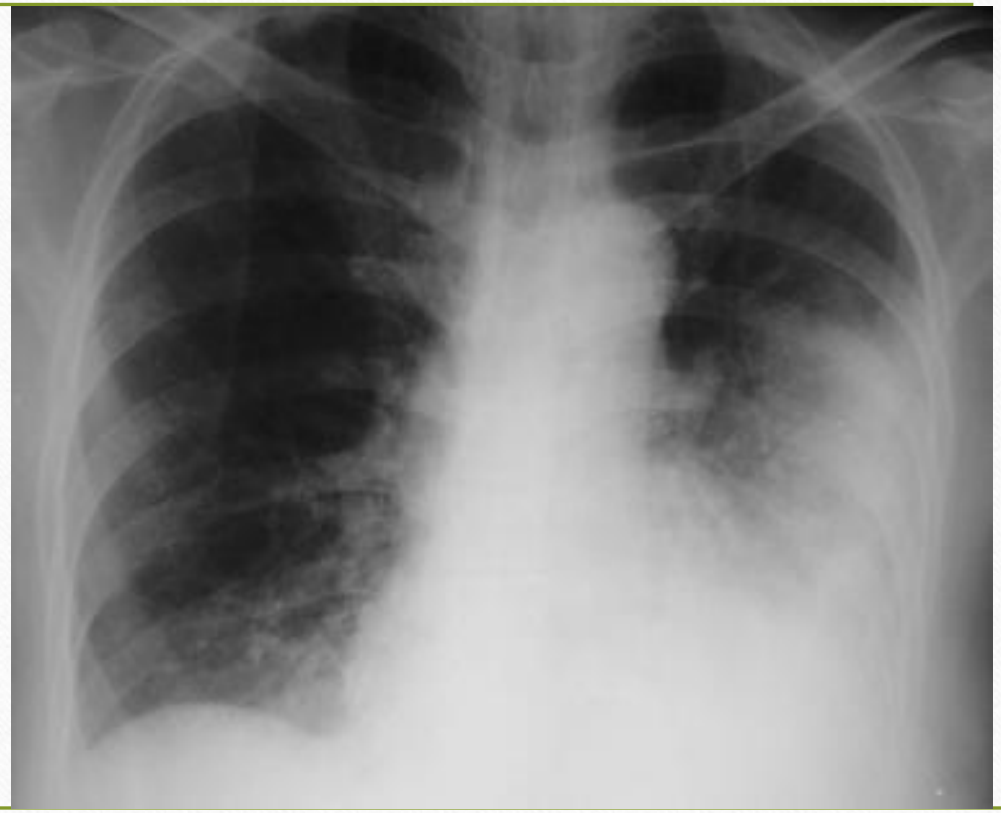
CHEST RADIOGRAPH

- This is PA film of RML pneumonia
- Note the indistinct borders, air bronchograms, and silhouetting of the right heart border.



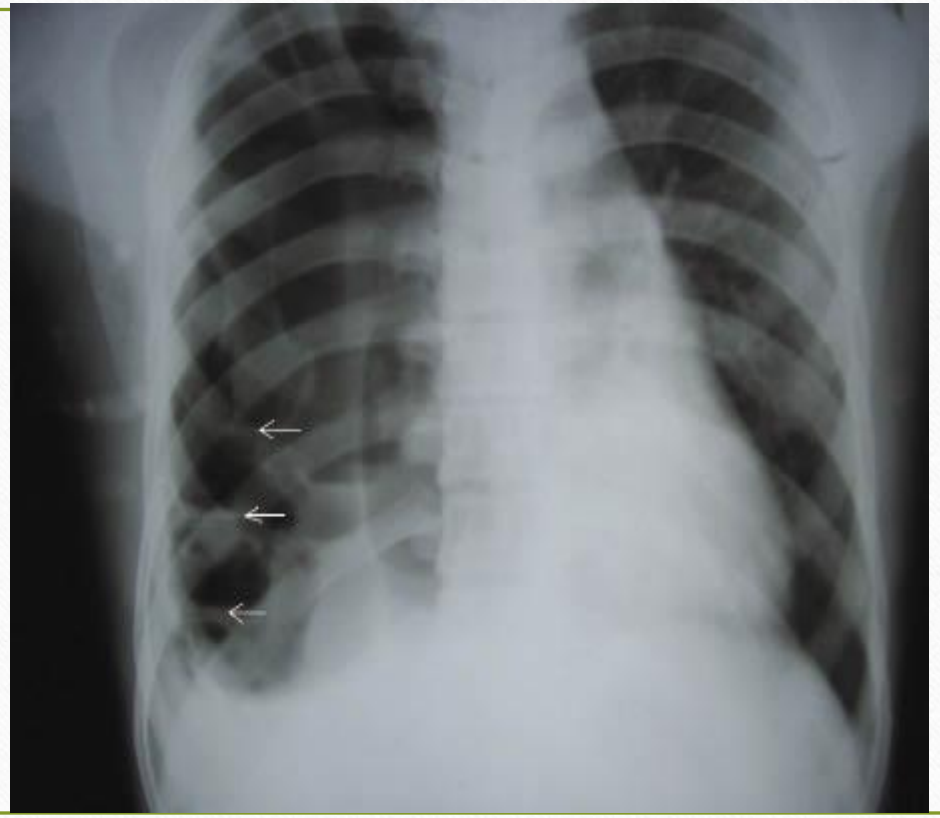
CHEST RADIOGRAPH

- The chest radiograph reveals a left lower lobe opacity with pleural effusion.



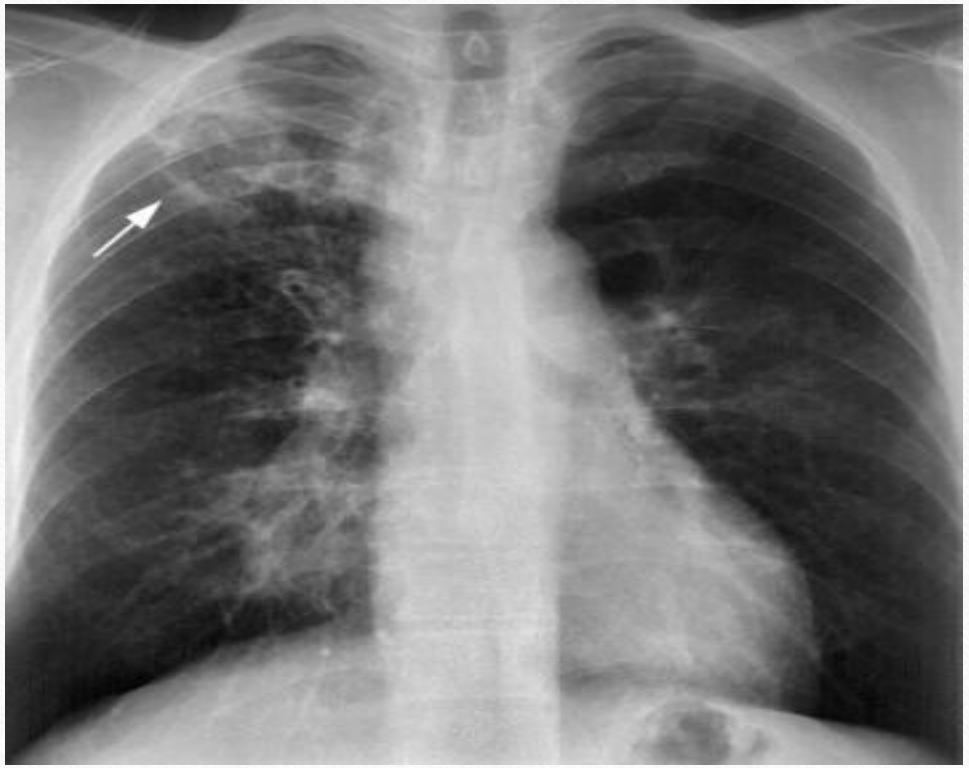
CHEST RADIOGRAPH

- White arrows indicating multiple pneumatocele



CHEST RADIOGRAPH

- Chest radiograph shows a prominent paratracheal area on the right, lymphadenopathy, a cavitary opacity in the right upper lobe, and a focal consolidation in the middle lung zone on the right



DIAGNOSIS

- Blood culture: high-risk patients—including those with neutropenia secondary to pneumonia, asplenia, complement deficiencies, chronic liver disease, or severe CAP
- PCR of nasopharyngeal swabs: respiratory viral infection, *Legionella* species, *M. pneumoniae*, *C. pneumoniae*, and mycobacteria

TREATMENT

Site of care:

- Tools that objectively assess the risk of adverse outcomes include:

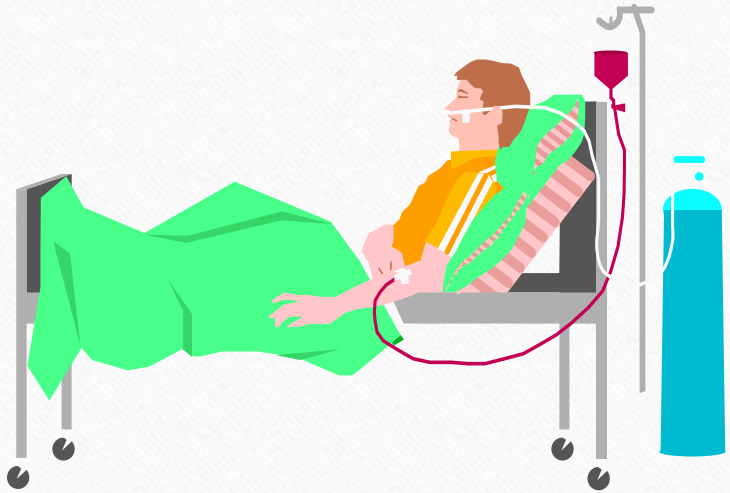
1) Pneumonia Severity Index (PSI), a prognostic model used to identify patients at low risk of dying; has 20 variables
2) CURB-65 criteria, a severity-of-illness score; five variables: confusion (C); urea >7 mmol/L (U); respiratory rate ≥30/min (R); blood pressure, systolic ≤90 mmHg or diastolic ≤60 mmHg (B); and age ≥65 years

RISK FACTORS FOR EARLY DETERIORATION

- Multilobar infiltrates
- Severe hypoxemia (arterial saturation <90%)
- Severe acidosis (pH <7.30)
- Mental confusion
- Severe tachypnoea (>30 breaths/min)
- Hypoalbuminemia
- Neutropenia
- Thrombocytopenia
- Hyponatremia
- Hypoglycemia

TREATMENT

- Adequacy of respiratory function
- Humidified oxygen for hypoxemia
- Bronchodilators (albuterol)
- Chest physiotherapy with postural drainage
- Adequate hydration if necessary
- Expectorants such as guaifenesin
- Chest pain- analgesics



TREATMENT

- Initial therapy is usually empirical, designed to cover the most likely pathogens

Outpatients

1. Previously healthy and no antibiotics in past 3 months

• A macrolide (clarithromycin [500 mg PO bid] or azithromycin [500 mg PO once, then 250 mg qd]) *or*

• Doxycycline (100 mg PO bid)

2. Comorbidities or antibiotics in past 3 months: select an alternative from a different class

• A respiratory fluoroquinolone (moxifloxacin [400 mg PO qd], gemifloxacin [320 mg PO qd], levofloxacin [750 mg PO qd]) *or*

• A β-lactam (preferred: high-dose amoxicillin [1 g tid] or amoxicillin/clavulanate [2 g bid]; alternatives: ceftriaxone [1–2 g IV qd], cefpodoxime [200 mg PO bid], cefuroxime [500 mg PO bid]) *plus* a macrolide

TREATMENT

Inpatients, Non-ICU

- A respiratory fluoroquinolone (e.g., moxifloxacin [400 mg PO or IV qd] or levofloxacin [750 mg PO or IV qd])
- A β-lactam (e.g., ceftriaxone [1–2 g IV qd], ampicillin [1–2 g IV q4–6h], cefotaxime [1–2 g IV q8h], ertapenem [1 g IV qd]) *plus* a macrolide (e.g., oral clarithromycin or azithromycin [as listed above] or IV azithromycin [1 g once, then 500 mg qd])

TREATMENT

Inpatients, ICU

• A β-lactam (e.g., ceftriaxone [2 g IV qd], ampicillin-sulbactam [2 g IV q8h], or cefotaxime [1–2 g IV q8h]) *plus* either azithromycin or a fluoroquinolone

If CA-MRSA is a consideration:

• Add linezolid (600 mg IV q12h) or vancomycin (15 mg/kg q12h initially, with adjusted doses)

TREATMENT

If Pseudomonas is a consideration:

- An antipseudomonal β-lactam (e.g., piperacillin/tazobactam [4. 5 g IV q6h], cefepime [1–2 g IV q12h], imipenem [500 mg IV q6h], meropenem [1 g IV q8h]) *plus* either ciprofloxacin (400 mg IV q12h) or levofloxacin (750 mg IV qd)
- The above β-lactams *plus* an aminoglycoside (amikacin [15 mg/kg qd] or tobramycin [1. 7 mg/kg qd]) *plus* azithromycin
- The above β-lactams *plus* an aminoglycoside *plus* an antipneumococcal fluoroquinolone

COMPLICATIONS

- Respiratory failure
- Shock
- Multiorgan failure
- Coagulopathy
- Exacerbation of comorbid illnesses
- Metastatic infection
- Lung abscess
- Complicated pleural effusion

PROGNOSIS

- Depends on the patient's age, comorbidities, and site of treatment (inpatient or outpatient)
- The overall mortality rate for the outpatient group is <1%
- For patients requiring hospitalization, the overall mortality rate is estimated at 10%, with ~50% of deaths directly attributable to pneumonia

PREVENTION

- Vaccinations available for Streptococcus pneumoniae and influenza virus
- Pneumococcal polysaccharide vaccine (PPV23): capsular material from 23 pneumococcal serotypes
- Protein conjugate pneumococcal vaccine (PCV13): capsular polysaccharide from 13 of the most frequent pneumococcal pathogens affecting children is linked to an immunogenic protein
- PCV13: decrease in the prevalence of antimicrobial-resistant pneumococci and in the incidence of invasive pneumococcal disease among both children and adults
- PCV13 now is also recommended for the elderly and for younger immunocompromised patients

PREVENTION

- Influenza vaccines:
- Intramuscular inactivated vaccine
- Intranasal live-attenuated cold-adapted vaccine: contraindicated in immunocompromised patients
- In the event of an influenza outbreak, unprotected patients at risk from complications should be vaccinated immediately and given chemoprophylaxis with either oseltamivir or zanamivir for 2 weeks

THANK YOU