Suppurative lung diseases
• Suppuration means pus formation.

• According to the site of pus formation, suppurative lung diseases (syndromes) will comprise;

1- Lung parenchyma (= lung abscess).
2- Bronchi (= bronchiectasis).
3- Pleura (= empyema).
Lung abscess
Definition;

Lung abscesses are pus-containing necrotic lesions of the lung parenchyma that often contain an air-fluid level. A similar process with multiple small cavities less than 2 cm in diameter has been designated necrotizing pneumonia by some clinicians.

Organisms;

the flora of upper respiratory tract, ~ 90% anaerobic
  • Out of hospital: anaerobes, pneumococci, staphylococci, enteric gram negatives (in elderly)
  • In hospital: both anaerobes and aerobes, usually S aureus and enteric gram negative bacilli
Categories:

1. Aspiratory lung abscess (Predisposing Conditions)
2. Secondary lung abscess (preexisting conditions)
3. Hematogenous lung abscess (Extrapulmonary infections)
Pathology:

- Lung abscess starts as an area of pneumonia
- Small zones of necrosis
- Coalesce together to form one or more large cavities of 1-2 cm
- Progression & enlargement to form the abscess cavity
- The abscess cavity well erode a bronchus
- Expectoration of purulent sputum with air fluid formation in the abscess cavity

Fate:
1. Complete cure (especially with good ttt)
2. Chronicity
3. Infection of the other lung
4. Open into pleura -> pyopneumothorax
5. Haematogenous spread
Clinical picture;

I- Of the underlying disease.
II- Of the lung abscess;

A- Symptoms:
1- General; fever, malaise, chills, anorexia, loss of wt. ....etc.
2- Local; cough, dyspnea, chest pain & tightness, haemoptysis and purulent sputum of large amounts.

B- Signs:
1- General; fever, tachypnea, tachycardia, pallor, clubbing ....etc.
2- Local; of pleurisy (rub), consolidation (br br, ↑ TVF), effusion ( dullness, ↓ intensity of breath sounds & ↓ TVF ).
Investigations;

I) Blood; CBC & ESR, blood culture & sensitivity and serology (for hydatid, amoeba.....etc.).

II) Radiology; CxR (PA & Lat), CT scan.

III) Microbiology; examination of:
1- Sputum (spont., or induced) for Gm & ZN stains, C&S (for aerobes, anaerobes & fungi + quantitative C).
2- Transtracheal aspirate.
3- Percut. needle aspiration.
4- PSB biopsy.

IV) F.O. Bronchoscopy; for PSB biopsy or to exclude the presence of FB or tumor
Differential diagnosis;

1. Pneumonia
   - Chest X-ray: infiltration without cavity
   - Short course
2. Pulmonary TB
   - Sputum smear for TB bacilli
   - Bronchoscopy
3. Bronchial carcinoma
   - Obstructive pneumonia
   - Cavitated bronchial carcinoma
4. Infected lung cyst
   - Chest X-ray: thin walled, prior radiograph
**Treatment**;

I- Antibiotics;
used in large doses, for sufficient time, parenteral, broadspectrum for aerobes and anaerobes ( gm +ve & gm –ve, cocci & bacilli ), and shift to oral antibiotics after that. Antibiotics of choice; penicillin, metronidazole, clindamycin and Others.

II- Drainage;
1- Postural drainage (physiotherapy).
2- Bronchoscopy, for drainage (tamponade, or removal of FB ).
3- Transthoracic needle aspiration (with toilet and local antibiotic injection).
III- Role of surgery; is very limited because antibiotics & drainage is effective in most cases.

A- Types of surgery;
1- Intercostal tube drainage or open drainage via pneumonostomy.
2- Resective surgery.

B- Indications;
1- Poor response to antibiotics.
2- Suspicion of lung cancer.
3- Massive or recurrent life threatening haemoptysis.
4- Complicating empyema.
Bronchiectasis
Definition;
Bronchiectasis is defined by permanent and abnormal widening of the bronchi.

Pathogenesis;
The most widely known model of the development of bronchiectasis is Cole’s “vicious cycle hypothesis”. In this model, Cole proposed that an environmental insult often on a background of genetic susceptibility impaired mucociliary clearance resulting in persistence of microbes in the sinobronchial tree and microbial colonization. The microbial infection caused chronic inflammation resulting in tissue damage and impaired mucociliary motility. In turn this led to more infection with a cycle of progressive inflammation causing lung damage.

The current view is that the two factors required for the development of this condition are persistent infection and a defect in host defense.
Etiologic/risk factors associated with bronchiectasis;

- Mucociliary disorder (immotile cilia, Kartagener’s syndrome, Young’s Syndrome, CF).
- Alpha1-antitrypsin deficiency.
- Postinfective (postpneumonia, whooping cough, measles, mycobacterial infection)
- Obstructive (foreign body, mycobacterial infection, obstructing cancer).
- Immune disorder (hypogammaglobulinemia, HIV infection, cancer, allergic bronchopulmonary aspergillosis, transplant rejection)
- Rheumatic/inflammatory disease (rheumatoid arthritis, inflammatory bowel disease)
- Extremes of age (immune system is less effective)
- Malnutrition/socioeconomic disadvantage
- Chronic obstructive pulmonary disease
- Aspiration
- Miscellaneous (yellow nail syndrome)
Pathology;

- Reid categorized bronchiectasis as having three main phenotypes:

1) tubular characterized by smooth dilation of the bronchi (the main form commonly seen);
2) varicose in which the bronchi are dilated with multiple indentations; and
3) cystic in which dilated bronchi terminate in blind ending

-Bronchial dilation is characterized by deficiency/loss of elastin and more advance disease by destruction of muscle and cartilage.
-Bronchial wall fibrosis, atelectasis and peribronchial pneumonic change.
HRCT examples of Reid’s three forms of bronchiectasis: A) tubular, B) varicose, and C) cystic.
**Distribution of bronchiectasis;**

Bronchiectasis has been described as being: localized (ie, confined to one lobe) or generalized.

- Most commonly it is generalized and seems to be most common in the lower lobes. The involvement of the lower lobes may reflect 
  gravity dependent retention of infected secretions.

- Right middle lobe bronchiectasis has been well described in the context of tuberculosis. The right middle lobe bronchus is long, often bends sharply at its bifurcation and is of relatively small caliber. A collar of lymph nodes also surrounds the proximal bronchus and any condition that causes a prolonged enlargement of these nodes may lead to obstruction and secondary bronchiectasis.
The classic clinical manifestations of bronchiectasis are;

- Daily cough and mucopurulent sputum production., frequently worse in the morning (having accumulated during recumbency in sleep) is present in most patients. Sputum production may be intermittent, being affected by recurrent infections, bronchial plugging, and antibiotic therapy.
- Hemoptysis may be seen in 40 to 70 percent of patients and may vary from blood streaks to large clots.
- Increasing cough, dyspnea, and volume of sputum production, fever, hemoptysis, and chest pain are hallmarks of acute exacerbations.
On physical examination;
I. Chest auscultation usually reveals findings of early and mid-inspiratory crackles as well as diffuse rhonchi and prolonged expiration. Bronchial breath sounds may be heard in severe cases or patients with a complicating pneumonia.
II. Digital clubbing and hypertrophic pulmonary osteoarthropathy,
III. In severe advanced cases, there may be evidence of respiratory insufficiency and cor pulmonale.
DIAGNOSIS:

- In the great majority of cases, bronchiectasis (BXSIS) is recognized in the context of chronic or recurring lower respiratory tract infections (deemed to be "bronchitis" or "pneumonia") over many months or years.
- Some patients in whom wheezing is a prominent element may have been identified and treated as "asthmatics" for many years.
- Occasionally patients come to attention following an episode of hemoptysis.
- Less frequently, BXSIS is identified on CT scans done for other considerations.
- Although the plain chest radiograph can suggest BXSIS with "tram tracks" or multiple ring shadows.

- CT scanning is the current diagnostic study of choice.

- The finding on the plain lateral chest radiographs of atelectasis of the right middle lobe and/or lingula is highly suggestive of coexisting BXSIS and should be followed by CT scanning in patients with persistent abnormalities and chronic symptoms.
High-Resolution Computed Tomography;

The HRCT has been proved to be a reliable and noninvasive method for assessment of bronchiectasis. (sensitivity of 97 percent) localize and describe areas of parenchymal abnormality, and identify bronchiolar abnormalities and mucus plugging to the level of fifth- and sixth-order bronchi.

It also can identify focal areas of air trapping as an indicator of small airway disease. Evidence of small airway plugging with debris (tree-in-bud) may also be seen.

**Airway dilatation can be detected by:**

- Finding tram lines or end-on-ring appearance.
- A luminal diameter more than 1.5 times the adjacent vessel is indicative of bronchiectasis.
- Bronchial wall thickening may also be seen.
Treatment;

- Postural drainage, and physiotherapy
- Symptomatic therapy.
- Antibiotics.
- Anti-inflammatory.
- Surgical (elective).
Cystic Fibrosis
Cystic fibrosis (CF) and its variants are a common cause of bronchiectasis. This is a monogenic disorder that presents most commonly in childhood as a multisystem disease. However, 3 to 7 % of patients with cystic fibrosis are diagnosed in adulthood, and due to improvements in therapy, 25 percent of childhood cases reach adulthood. This is an autosomal recessive condition resulting from a genetic defect located on chromosome 7 leading to a deficiency in the CF transmembrane regulator. An inherited disorder that causes severe damage to the lungs and digestive system.
It affects mostly the lungs but also the pancreas, liver, kidneys, and intestine.

Cystic fibrosis affects the cells that produce mucus, sweat and digestive juices.

The secretions to become thick and sticky. Instead of acting as a lubricant, the secretions plug up tubes, ducts and passageways, especially in the lungs and pancreas.
Respiratory signs and symptoms

• A persistent cough that produces thick (sputum) mucus
• Wheezing
• Breathlessness
• Exercise intolerance
• Repeated lung infections
• Inflamed nasal passages or a stuffy nose

Digestive signs and symptoms

• Foul-smelling, greasy stools
• Poor weight gain and growth
• Intestinal blockage, particularly in newborns (meconium ileus)
• Severe constipation
• +/- Diabetes Mellitus
Complications

- Bronchiectasis
- Nasal polyps
- Hemoptysis
- Pneumothorax
- Respiratory failure
- Nutritional deficiencies.
- Diabetes
- Blocked bile duct
- Intestinal obstruction
- Distal intestinal obstruction syndrome
- Infertility
- Osteoporosis
- Electrolyte imbalances and dehydration
Diagnosis

- Genetic testing
- **Sweat test**: concentration of chloride is greater than or equal to 60 mmol/L
- Chest radiology
- Tests for pancreatic enzymes
Treatments

The goals of treatment include:
• Preventing and controlling lung infections
• Loosening and removing mucus from the lungs
• Preventing and treating intestinal blockage
• Providing adequate nutrition

1- Medications:

• Antibiotics
• Mucuolytic
• Bronchodilators
• Oral pancreatic enzymes to help your digestive tract absorb nutrients
2- Chest physical therapy: A common technique is clapping with cupped hands on the front and back of the chest. (Mechanical devices also can help)

3- Surgical and other procedures:

- Nasal polyp removal
- Oxygen therapy
- Bronchoscopy and lavage
- Bowel surgery
- Lung transplant
EMPYEMA
**Definition:**
Empyema is defined by the presence of pus in the pleural space.

**Aetiology:**
- Direct extension of a pulmonary parenchymal infection into the Pleural space causes more than half the cases of empyema.
- Postsurgical infection accounts for an additional 20 percent.
- Penetrating or blunt trauma to the thorax.
- Sometimes, bacteria from abdominal infection, such as a subdiaphragmatic abscess, cross the diaphragm and enter the pleural space.
The bacteriology of empyema;

- Anaerobic organisms are now isolated from up to 75 percent of patients.

- Aerobic gram-negative rods have increased markedly: *Haemophilus* spp., *Klebsiella* spp., *E. coli* and *Enterobacter* spp.

- *S. aureus*, *S. pneumoniae* and *S. pyogenes*
Clinical manifestations;

- Those of the underlying disease.

- Physical examination indicates the presence of pleural fluid, with dullness to percussion, decreased breath sounds, egophony, and diminished tactile fremitus on the side of involvement.

- Mediastinal shift may occur.
Investigations;

I. Chest radiographs:
- PA (postero-anterior) with lateral decubitus views may be needed to detect small volumes of fluid (less than 250 mL).
- CT scans can distinguish loculations, identify intrapulmonary abnormalities such as a lung abscess or tumor, detect bronchopleural fistulas, and verify the position of chest tubes.

II. Diagnostic aspiration:
- Pleural fluid that appears as gross pus or in which large numbers of bacteria are seen on Gram stain is an empyema.
- Otherwise, the pH (below 7.2) and glucose content (less than 40-60 mg/dl) of pleural fluid differentiate empyema from benign parapneumonic effusion and noninfectious causes of exudate.
Treatment;
2. Vigorous pleural drainage;
   - In the early exudative phase of empyema formation, repeated thoracentesis may provide adequate drainage.
   - More typically, the fluid is viscous and reaccumulates rapidly, requiring closed chest tube drainage. (under ultrasound or CT guidance).
3. Video-assisted thorascopic surgery may obviate the need for open drainage with rib resection.
4. Decortication of the empyema sac for organized fibrotic empyema.
5. Primary site of infection should be eradicated. If present, concomitant subphrenic or mediastinal abscesses require surgical intervention.
Criteria for Tube Thoracostomy in Parapneumonic Effusions and Empyema;

**Radiographic criteria:**
- Pleural fluid loculations
- Effusion filling more than half the hemithorax
- Air-fluid level

**Microbiologic criteria:**
- Pus in the pleural space
- Positive stain for microorganisms
- Positive pleural fluid cultures

**Chemical criteria:**
- Pleural fluid pH < 7.2
- Pleural fluid glucose < 60 mg/dl
Thank You