COPD and Bronchiectasis

Objectives:

- Definition of COPD and Bronchiectasis.
- Clinical and radiological diagnosis.
- Differential diagnosis.
- General outline of management.
- Create a link to 341 clinical teaching.

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- Editing file
- Feedback

Color index: IMPORTANT - NOTES - EXTRA - Books

Introduction to COPD

Definition :

- Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

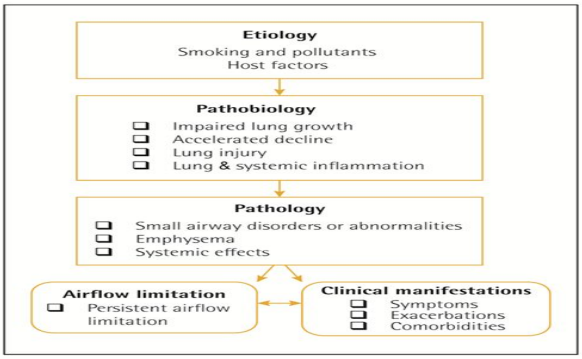
- Characterized by respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.

- Related diagnoses include **1.Emphysema** (pink puffers) and **2.Chronic Bronchitis** (blue bloaters):

- They are grouped together under COPD because they are both mainly caused by smoking (tobacco destroys elastin fibers)1 and they usually present together.
- Pure emphysema or pure chronic bronchitis are rare.

- The main risk factor for COPD is tobacco smoking but other environmental exposures such as biomass **"food cooked close to fire smoke"** fuel exposure and air pollution may contribute.

Figure 1.1. Etiology, pathobiology and pathology of COPD leading to airflow limitation and clinical manifestations



Epidemiology and etiology:

Prevalence of COPD was higher in smokers and ex-smokers compared to non-smokers

Higher ≥ 40 year group compared to those < 40 **"young ages who get COPD , their lungs deteriorate faster"**

Higher in men than women

Estimated 384 million COPD cases in 2010.

Three million deaths annually. By 2030 predicted 4.5 million COPD related deaths annually.

(long exposure to smoking leads to COPD and if the patient still smokes that could lead to CVD which leads to death)

Chronic bronchitis: Characterized by chronic cough productive of sputum for at least 3 months per year for at least 2 consecutive years.

Emphysema: Characterized by permanent enlargement of air spaces distal to terminal bronchioles due to destruction of alveolar walls. It is classified into :

Centri-acinar : Common in smokers,the damage happens in proximal acini.

Pan-acinar: in patient with deficiency of alpha 1 antitrypsin, the damage happens in both proximal & distal acini

Irregular: in patient with chronic inflammation of the lung (ex: TB) , the damage happens in many area.

- ## Alpha-1 antitrypsin deficiency (AATD)

- The World Health Organization recommends that all patients with a diagnosis of COPD should be screened once especially in areas with high AATD prevalence.
- AATD patients are typically < 45 years with panlobular basal emphysema
- Delay in diagnosis in older AATD patients presents as more typical distribution of emphysema (centrilobular apical).
- A low concentration (< 20% normal) is highly suggestive of homozygous deficiency.

Pathophysiology and Risk Factors of COPD

COPD is characterized by structural changes and chronic inflammation that lead to :

1. Airflow limitation and gas trapping.
2. Gas exchange abnormalities.
3. Mucus hypersecretion.
4. Pulmonary hypertension.

- ## Pathogenesis:

Chronic irritation of airways (ex:Smoking or α1-antitrypsin deficiency) → chronic inflammation and cells produce inflammatory mediators → oxidative stress → protease and antiprotease imbalance → Narrowing of airways and Peribronchiolar and interstitial fibrosis → structural changes.

- Smoking for long time leads to accumulation of oxidative stress which inhibits Antiprotease that leads to Protease-Antiprotease imbalance

Because of these narrowing and structural changes, COPD patient will present with different symptoms.

Very helpful Summary here.

Risk factors:

1. Tobacco smoke (in 90% of COPD cases).
2. α1-antitrypsin deficiency (risk is worse in combination with smoking).
3. Environmental factors e.g. second hand smoking.
4. Chronic asthma.
5. Others. (see the chart)

19.27 Risk factors for development of COPD

Environmental

- Tobacco smoke accounts for 95% of cases in UK
- Indoor air pollution; cooking with biomass fuels in confined areas in developing countries
- Occupational exposures, such as coal dust, silica and cadmium
- Low birth weight may reduce maximally attained lung function in young adult life
- Lung growth: childhood infections or maternal smoking may affect growth of lung during childhood, resulting in a lower maximally attained lung function in adult life
- Infections: recurrent infection may accelerate decline in FEV_1; persistence of adenovirus in lung tissue may alter local inflammatory response, predisposing to lung damage; HIV infection is associated with emphysema
- Low socioeconomic status
- Cannabis smoking

Host factors

- Genetic factors: α_1-antiproteinase deficiency; other COPD susceptibility genes are likely to be identified
- Airway hyper-reactivity

The Clinical Features of COPD

Clinical features:

Symptoms	Signs
1. Chronic and progressive dyspnea 2. Cough 3. Sputum production 4. Wheezing and chest tightness. 5. Others – including fatigue, weight loss, anorexia, syncope, rib fractures, ankle swelling, depression, anxiety.	**Signs—the following may be present:** • Prolonged expiratory time. • During auscultation, end-expiratory wheezes on forced expiration, decreased breath sounds, and/or inspiratory crackles. • Tachypnea, tachycardia. • Cyanosis. • Use of accessory respiratory muscles. • Hyperresonance on percussion. • Signs of cor pulmonale.

why you have (cough syncope)? due to valsava maneuver

- Finger clubbing is not a feature of COPD and should trigger further investigation for lung cancer, bronchiectasis or fibrosis.

Table 2.2. Other causes of chronic cough

Intrathoracic	Extrathoracic
• Asthma	• Chronic allergic rhinitis
• Lung cancer	• Post nasal drip syndrome (PNDS)
• Tuberculosis	• Upper Airway Cough Syndrome (UACS)
• Bronchiectasis	• Gastroesophageal reflux
• Left heart failure	• Medication (e.g. ACE inhibitors)
• Interstitial lung disease	
• Cystic fibrosis	
• Idiopathic cough	

How to differentiate between:

chronic bronchitis, emphysema, and COPD

1. **Chronic bronchitis**: is a clinical diagnosis of increased sputum over a period of time,
2. **Emphysema**; is a pathological diagnosis which is defined by loss of alveolar capillaries
3. **COPD:** is a clinical syndrome which requires a history of smoking, wheezing, and spirometry (decreased FEV1/FVC ratio)

Asthma and COPD

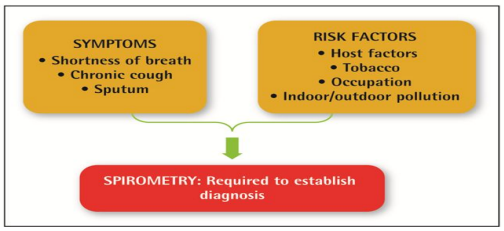
1. Age of onset
2. History of atopy: present with asthma.
3. Eosinophilia and IgE: present with atopic asthma.
4. Bullae with COPD not with asthma
5. Low FVC (with both asthma & COPD)
6. Chronic respiratory failure: chronic obstruction of the lung (with COPD) may lead to respiratory failure. While asthma doesn't cause respiratory failure (yes asthma cause dyspnea during the attack but not causing respiratory failure or damage to alveoli)
7. Diffusing capacity: in asthma is normal bc the alveoli is not involved
8. Trial of inhaled corticosteroids: in atopic asthma if u give corticosteroid it will abolished the air way obstruction. While COPD has poor response to corticosteroid.

Extra

Diagnosis and Investigations of COPD

- Investigations:

Figure 2.1. Pathways to the diagnosis of COPD



1. Pulmonary function testing (Spirometry): (definitive diagnostic test)

• Decreased FEV1 and decreased FEV1/FVC ratio.

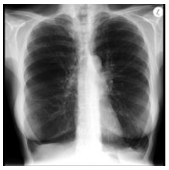
Table 2.4. Classification of airflow limitation severity in COPD (Based on post-bronchodilator FEV_1)		
In patients with $FEV_1/FVC < 0.70$:		
GOLD 1:	Mild	$FEV_1 \geq 80\%$ predicted
GOLD 2:	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3:	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4:	Very Severe	$FEV_1 < 30\%$ predicted

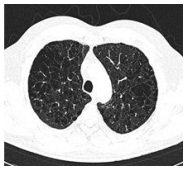
2. Chest radiograph (CXR)

Low sensitivity for COPD; only severe, advanced emphysema will show the typical changes, which include:

• Hyperinflation (Barrel Chest with descending of the liver) , flattened diaphragm. Diminished vascular markings.

Useful in an acute exacerbation to rule out complications such as pneumonia or pneumothorax.





3. Measure α1-antitrypsin levels in patients with a personal or family history of premature emphysema (≤50 years old).

4. Arterial blood gas (ABG) : chronic PCO2 retention, decreased PO2.

Initially PaCO2 normal but later on will be hypercapnia (because of bronchitis no enough air getting out)

retention of CO2 → respiratory ACIDOSIS (↑CO2 & low PH) → as compensatory mechanism HCO3 will be high

In order to diagnose COPD we have to rule out other diseases that causes the same symptoms as COPD like cancers, GERD and Heart Failure

Choice of thresholds:

- COPD Assessment Test (CAT TM).
- Modified Medical Research Council (mMRC) questionnaire.

Figure 2.3. CAT Assessment

For each item below, place a mark (X) in the box that best describes you currently. Be sure to only select one response for each question.

Example: I am very happy 0 (X) 2 3 4 5 I am very sad — SCORE

I never cough	0 1 2 3 4 5	I cough all the time
I have no phlegm (mucus) in my chest at all	0 1 2 3 4 5	My chest is completely full of phlegm (mucus)
My chest does not feel tight at all	0 1 2 3 4 5	My chest feels very tight
When I walk up a hill or one flight of stairs I am not breathless	0 1 2 3 4 5	When I walk up a hill or one flight of stairs I am very breathless
I am not limited doing any activities at home	0 1 2 3 4 5	I am very limited doing activities at home
I am confident leaving my home despite my lung condition	0 1 2 3 4 5	I am not at all confident leaving my home because of my lung condition
I sleep soundly	0 1 2 3 4 5	I don't sleep soundly because of my lung condition
I have lots of energy	0 1 2 3 4 5	I have no energy at all

TOTAL SCORE

Reference: Jones et al. ERJ 2009; 34 (3): 648-54.

Table 2.5. Modified MRC dyspnea scale[a]

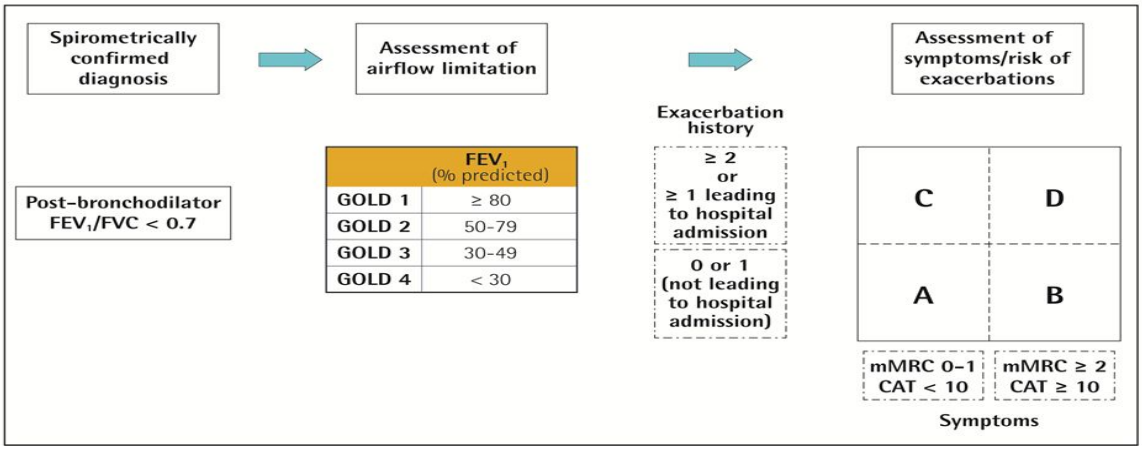
PLEASE TICK IN THE BOX THAT APPLIES TO YOU (ONE BOX ONLY) (Grades 0-4)	
mMRC Grade 0. I only get breathless with strenuous exercise.	☐
mMRC Grade 1. I get short of breath when hurrying on the level or walking up a slight hill.	☐
mMRC Grade 2. I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level.	☐
mMRC Grade 3. I stop for breath after walking about 100 meters or after a few minutes on the level.	☐
mMRC Grade 4. I am too breathless to leave the house or I am breathless when dressing or undressing.	☐

[a] Fletcher CM. BMJ 1960; 2: 1662.

❖ **Based on the assessment tools, the physician will determine the management.**

- **ABCD Assessment Tool:**

Figure 2.4. The refined ABCD assessment tool

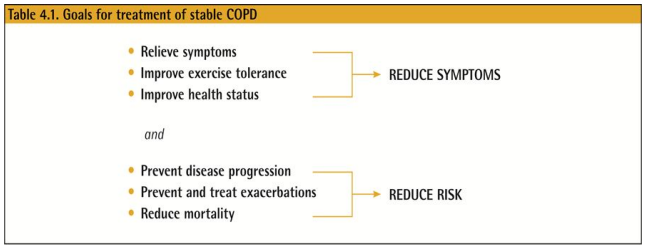


-exacerbation history:
how many times last (year) have you been admitted to hospital for exacerbation?

-Assessment of symptoms / risk of exacerbation:
Group A: ↓exacerbation , ↓symptoms
Group B: ↓exacerbation , ↑ symptoms
Group C: ↑exacerbation , ↓ symptoms
Group D: ↑exacerbation , ↑ symptoms

Management of COPD

Once COPD has been diagnosed, effective management should be based on an individualized assessment to reduce both current symptoms and future risks of exacerbation.



○ **Improve mortality and delays progression of disease:**	**1. Smoking cessation** **The most important one**	• Smoking cessation has the greatest capacity to influence the natural history of COPD. • If effective resources and time are dedicated to smoking cessation, long-term quit success rates of up to 25% can be achieved[1].
	2. Long term oxygen therapy (LTOT) here	Long-term oxygen therapy is indicated for stable patients who have: • PaO2 at or below 7.3 kPa (55 mmHg) or SaO2 at or below 88%, with or without hypercapnia confirmed twice over a three week period; or • PaO2 between 7.3 kPa (55 mmHg) and 8.0 kPa (60 mmHg), or SaO2 of 88%, if there is evidence of pulmonary hypertension, peripheral edema suggesting congestive cardiac failure, or polycythemia (hematocrit > 55%). - Only in patients with hypoxia/cor pulmonale
	3. Vaccinations In order to prevent infections that could lead to exacerbation	• Influenza vaccination can reduce serious illness (such as lower respiratory tract infections requiring hospitalization)24 and death in COPD patients. • Pneumococcal vaccinations, PCV13 and PPSV23, are recommended for all patients ≥ 65 years of age.
○ **Improves symptoms (But does not decrease disease progression or mortality):**	**4. Bronchodilators[2] (Inhaled)** to relieve the symptoms	a. Short acting bronchodilators for **mild** disease - **Inhaled Beta 2 agonists:** Salbutamol, Terbutaline **salbutamol causes hypokalemia** b. Long acting bronchodilators for **moderate** to **severe** disease. c. Inhaled Anticholinergics[3] (muscarinic antagonists) are more appropriate for patients with **moderate** to **severe** disease as: Tiotropium bromide, Ipratropium bromide.

[1] (This initial inflammation of the small airways is reversible and accounts for the improvement in airway function if smoking is stopped early.)
[2] minimal effect. Improve FEV1 by 5-10%
[3] Inhaled anticholinergic agents are most effective in COPD

cont.. **Improves symptoms**		*This is because it is believed that beta agonists might cause CVS side effects (tachycardia. etc.) They also have slower onset of action than beta blockers but last longer. - Combination of beta agonist Salbutamol "albuterol" with ipratropium bromide is more efficacious than either agent alone in bronchodilation. Also help in adherence to therapy "both medications in one inhaler"
	Cont… Bronchodilators (Oral)	Can be given to patients who ***cannot*** inhale efficiently. ❖ **Theophylline:** - not commonly used because it has a lot of side effects and drug interactions + Narrow therapeutic index, so serum levels must be monitored. - Improves mucociliary clearance and central respiratory drive.
	5. Corticosteroids[4]	a. **Inhaled corticosteroids** (anti-inflammatory) Usually given in combination with **long acting beta agonists.**[5] b. **Oral corticosteroids** are useful in acute exacerbations (discussed later) "systemic glucocorticoids are only used for acute exacerbations and **should not** be used for long-term treatment, even for pts with severe COPD" - Remember steroids have many side effects such as osteoporosis and Cushing's syndrome.
	6. Pulmonary Rehabilitation	Education, exercise, physiotherapy: A major goal is to improve exercise tolerance. Pulmonary rehabilitation improves functional status and quality of life. • **Exercise training:** 6–12 weeks; longer programs result in larger effects 20–30 min walking per session, to limits of symptoms • **Patient education:** Smoking cessation, COPD natural history and management, self-management, exacerbations • **Assessment and follow-up** • **Nutritional support**
	7. Surgery:	May be beneficial in selected patients; carefully weigh potential benefits with risks. Options include: • Surgical or endoscopic Lung resection (LVRS) **"removing part of the lung that interferes with normal function"** • Bullectomy • Lung transplantation

❖ See the table in next slide to understand more

[4] In symptomatic patients with moderate/severe COPD, a trial of corticosteroids is always indicated.
[5] Combination of a corticosteroid with a long-acting p2 agonist has been shown in a trial to protect against a decline in lung function but there was no reduction in overall mortality

❖ **This table will show you when to use the medications above:**

Treatment guidelines	
Mild to **moderate** disease	**Severe** disease
1. **Bronchodilator** in an inhaler (use spacer to improve delivery), anticholinergic drugs and/or beta agonists are first-line agents. 2. Inhaled **corticosteroids** may be used as well. Use lowest dose possible 3. Theophylline may be considered if the above do not adequately control symptoms.	1. Medications as mild-moderate. 2. Continuous **oxygen** therapy if patient is hypoxemic 3. **Pulmonary rehabilitation.** 4. **Triple inhaler therapy** (long acting beta agonist + long acting anticholinergic + an inhaled glucocorticoid)

Complications of COPD

- **Acute exacerbations** (mostly due to infections or noncompliance). **"exacerbation= worsen of symptoms"**
- Respiratory failure (COPD is the most common cause)
- Cor pulmonale (Hypoxia induces constriction of pulmonary arterioles →increase pulmonary resistance → result in pulmonary hypertension and right ventricular hypertrophy with or without right sided heart failure)
- Pulmonary HTN (constriction of pulmonary arterioles due to hypoxia)
- Others : Bacterial colonisation, Secondary polycythemia, Hemoptysis Pneumothorax (not very common)

- **Acute exacerbation of COPD[6] :**

COPD exacerbations are defined as an acute worsening of respiratory symptoms that result in additional therapy. They are classified as:

A. **Mild :** (treated with short acting bronchodilators only, SABDs (Short acting bronchodilators))
B. **Moderate :** (treated with SABDs plus antibiotics and/or oral corticosteroids)
C. **Severe :** (patient requires hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure.

- **Classification of hospitalized patients**

No respiratory failure:

Respiratory rate: 20-30 breaths per minute; no use of accessory respiratory muscles; no changes in mental status; hypoxemia improved with supplemental oxygen given via Venturi mask 28-35% inspired oxygen (FiO2); no increase in PaCO2.

Acute respiratory failure — non-life-threatening:

Respiratory rate: > 30 breaths per minute; using accessory respiratory muscles; no change in mental status; hypoxemia improved with supplemental oxygen via Venturi mask 25-30% FiO2; hypercarbia i.e., PaCO2 increased compared with baseline or elevated 50-60 mmHg

Acute respiratory failure — life-threatening:

Respiratory rate: > 30 breaths per minute; using accessory respiratory muscles; acute changes in mental status; hypoxemia not improved with supplemental oxygen via Venturi mask or requiring FiO2 > 40%; hypercarbia i.e., PaCO2 increased compared with baseline or elevated > 60 mmHg or the presence of acidosis (pH < 7.25).

[6]They may be accompanied by the development of respiratory failure and/ or fluid retention.

Management of acute exacerbation of COPD:

A. Bronchodilators (β 2-agonist) alone or in combination with anticholinergics are first-line therapy.

B. Systemic corticosteroids are used for patients requiring hospitalization (IV methylprednisolone is a common choice). Do not use inhaled corticosteroids in acute exacerbations.

C. Antibiotics (azithromycin, levofloxacin, doxycycline, etc.)

D. Supplemental oxygen is used to keep O2 saturation 90% to 93%. Start with a nasal cannula; a face mask may need to be used.

Table 5.4. Indications for respiratory or medical intensive care unit admission*

- Severe dyspnea that responds inadequately to initial emergency therapy.
- Changes in mental status (confusion, lethargy, coma).
- Persistent or worsening hypoxemia (PaO_2 < 5.3 kPa or 40 mmHg) and/or severe/worsening respiratory acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation.
- Need for invasive mechanical ventilation.
- Hemodynamic instability–need for vasopressors.

*Local resources need to be considered.

Table 5.5. Indications for noninvasive mechanical ventilation (NIV)

At least one of the following:

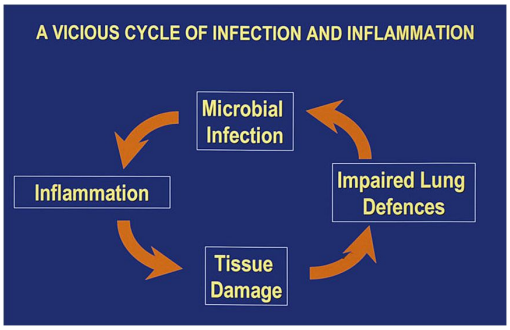
- Respiratory acidosis ($PaCO_2$ ≥ 6.0 kPa or 45 mmHg and arterial pH ≤ 7.35).
- Severe dyspnea with clinical signs suggestive of respiratory muscle fatigue, increased work of breathing, or both, such as use of respiratory accessory muscles, paradoxical motion of the abdomen, or retraction of the intercostal spaces.
- Persistent hypoxemia despite supplemental oxygen therapy.

Bronchiectasis

Bronchiectasis is characterized by permanent abnormal dilatation with impaired mucociliary clearance and excessive airways inflammation. It could lead to bacterial colonisation and infection. Bronchiectasis is uncommon because of better control of infections of the lung which lead to the weakening of the bronchial walls.

Pathogenesis:

There is permanent, abnormal dilation and destruction of bronchial walls with chronic inflammation, airway collapse, and ciliary loss/dysfunction leading to impaired clearance of secretion[7].



➔ Dilation of the airways prone to infection → infection → inflammation → tissue damage and ultimately impaired lungs defences which lead to infections again …

[7] The bronchiectatic cavities may be lined by granulation tissue, squamous epithelium or normal ciliated epithelium. There may also be inflammatory changes in the deeper layers of the bronchial wall and hypertrophy of the bronchial arteries.

Causes of bronchiectasis :

Cause	n (% of study)
Post infection	51 (32)
Idiopathic	42 (26)
PCD	17 (11)
ABPA	13 (8)
Immune deficiency	9 (6)
Ulcerative colitis	5 (3)
Young's syndrome	5 (3)
Pan bronchiolitis	4 (3)
Yellow nail syndrome	4 (3)
Mycobacterium infection	4 (3)
Rheumatoid arthritis	3 (2)
Aspiration	2 (1)
CF variant	2 (1)
Total	161

1. **Acquired bronchiectasis:**

- Recurrent pulmonary infection (aspergillosis, H. influenzae, measles, **mycobacterium tuberculosis**, Staph. Aureus, P. aeruginosa).
- Bronchial obstruction
- Childhood infection e.g measles, pertussis
- Aspiration

2. **Congenital bronchiectasis:**

- Kartagener's syndrome (Ciliary dyskinesia → impaired mucociliary clearance → infection)
- Hypogammaglobulinemia (Abnormal lung defence → infection)
- Cystic fibrosis (Most common cause, accounts for half of all cases)

  (Thick mucus → increase the likelihood of infections → bronchiectasis)
- Abnormal cartilage formation.

Clinical features:

- Mainly similar to COPD, with increased likelihood for infections **commonly pneumonias** "recurrent or persistent pneumonia"
  (because the blocked area is an excellent place for bacteria to grow)
- **Chronic cough with large amount of mucopurulent copious foul smelling sputum.** (Due to infection)**to assess the amount of sputum produced, we compare it to فنجال**
- **Hemoptysis** due to rupture of blood vessels near bronchial wall surface.
  May be life threatening
- **Dyspnea, Weight loss and Fever**.
- **Clubbing (doesn't come in COPD or Asthma)**

19.37 Symptoms of bronchiectasis

- **Cough**: chronic, daily, persistent
- **Sputum**: copious, continuously purulent
- **Pleuritic pain**: when infection spreads to involve pleura, or with segmental collapse due to retained secretions
- **Haemoptysis**:
  - Streaks of blood common, larger volumes with exacerbations of infection
  - Massive haemoptysis requiring bronchial artery embolisation sometimes occurs
- **Infective exacerbation**: increased sputum volume with fever, malaise, anorexia
- **Halitosis**: frequently accompanies purulent sputum
- **General debility**: difficulty maintaining weight, anorexia, exertional breathlessness

❖ Bronchiectasis patients usually are:

- young age at presentation .
- symptoms over many years.
- absence of smoking history.
- daily expectoration of large volumes of sputum.
- hemoptysis.

❖ History which should lead to suspicion of bronchiectasis:

- Recurrent LRTI.
- Chronic productive cough.
- Breathlessness, wheeze.
- haemoptysis.
- Chest pain
- Tiredness
- (ENT,infertility,GI ,ILD) (patients with Kartagener's syndrome usually are infertile and deafs)

Thought to have COPD:

- COPD with bronchiectasis.
- no history of smoking
- there is slow recovery from lower respiratory tract infections
- recurrent exacerbations
- sputum growth/colonised with Pseudomonas aeruginosa

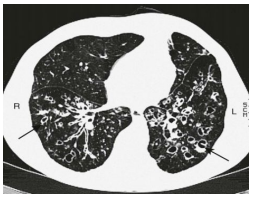
Investigations:

1. **Culture** patient's sputum:

because they often have special infections (Pseudomonas aeruginosa) and we should know their antibiotic sensitivity by culture in order to properly treat it.

2. **HR-CT scan:** [8] (the best non-invasive test)

shows thickened dilated bronchi "most accurate test, study of choice."



3. **Chest x-ray (CXR):**

It might be normal BUT in advanced cases it may show dilated bronchi with thickened bronchial walls and sometimes multiple cysts containing fluid and crowding of bronchi (tram tracking).

4. **Spirometry :** PFTs reveals an obstructive pattern.

5. Can also look for diseases that cause this condition For example: screen for ciliary dysfunction, CF, etc.

6. Bronchoscopy applies in certain cases.

Management of Bronchiectasis

1. **Chest physiotherapy :** (postural drainage, chest percussion) to help remove the mucus.

2. **Immunization :** In order to prevent infections that could lead to exacerbation.

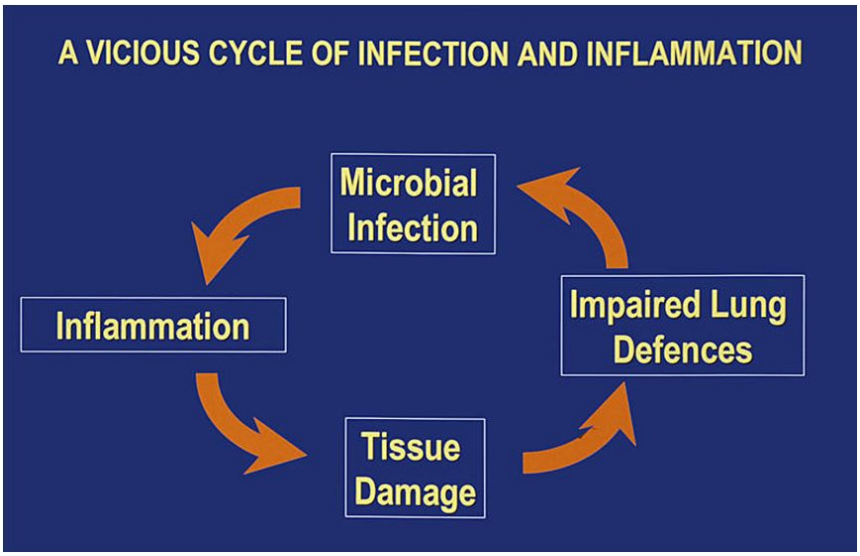
3. **Inhaled bronchodilators:** (If the patient has airflow obstruction).

4. **Mucolytics.** (to remove the mucus)

5. **Nebulised saline** (Hydration of airways → improve the respiration)

[8] with a sensitivity of 97%.

❖ **Our aim is to interrupt this cycle to prevent further infection and further damage**



- **Exacerbation of bronchiectasis characterized by :**

1.Deterioration over days

2.Increasing Cough

3.Increased sputum volume or change of viscosity

4.Increased sputum purulence + increasing wheeze & breathlessness

5.Haemoptysis

6.Systemic upset.

- **Empiric Therapy :**

- Amoxicillin 500mg tds[9] 14 days.
- Clarithromycin 500 bd[10]
- Severe Bronchiectasis/colonised with H influenzae : Amoxicillin 1g tds/ 3g bd

- Pseudomonas colonised patients Ciprofloxacin 500\750 bd

- **Antibiotics for acute exacerbations:**

Streptococcus pneumoniae

Amoxicillin 500 mg tds / Clarithromycin 500 mg bd 14 days.

[9] tds: ter die sumendum (three times a day)

[10] bd: bis die sumendum (two times a day)

Haemophilus influenzae (b-lactamase negative)
Amoxicillin 500 mg tds / Amoxicillin 1 g tds / Amoxicillin 3 g bd / Clarithromycin 500 mg bd.

Haemophilus influenzae (b-lactamase positive)
Co-amoxiclav 625 mg tds / Clarithromycin 500 mg bd / Ciprofloxacin 500 mg bd

Moraxella catarrhalis
Co-amoxiclav 625 mg tds / Ciprofloxacin 500 mg bd.

Staphylococcus aureus (MSSA)
Flucloxacillin 500 mg qds / Clarithromycin 500 mg bd.

Staphylococcus aureus MRSA
Coliforms / Ciprofloxacin.

When to give long term Antibiotics ?

- More than 3 Exacerbations/yr.
- Fewer Exacerbation in patients with significant morbidity.

Nebulised antibiotics Gent/tobramycin/colistin or Long term Macrolides.

"usually given in high doses due to damage of the lung (for 2 weeks)"

When to admit patients because of bronchiectasis ?

- Development of cyanosis or confusion
- Breathlessness with a respiratory rate >25/minute
- Circulatory failure, respiratory failure, cyanosis or confusion
- Temperature >38°C
- Patient unable to take oral therapy
- Patient unable to cope at home
- Haemoptysis >25mls/day
- Intravenous therapy required in patients with clinical failure after oral antibiotics

Monitoring patients with bronchiectasis:

- Symptoms.
- Sputum Volume 24hrs/Purulence.
- Frequency of Exacerbations/yr.
- Frequency of Antibiotic use.
- FEV1/ FVC annually.
- CXR only if indicated.

Summary

<table>
<tr><td colspan="3">COPD:
• a disease state characterized by persistent airflow limitation that is not fully reversible.
• The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.</td></tr>
<tr><td rowspan="4">Two types
(that usually coexist together)</td><td rowspan="2">Chronic bronchitis
“blue bloaters”</td><td>Definition:
Defined by clinical features: chronic productive cough for ≥ 3 months/year for at least 2 successive years.</td></tr>
<tr><td>Pathogenesis:
Airflow limitation due to:
• Excess mucus production.
• Inflammation & scarring in airways and enlargement in mucus glands.</td></tr>
<tr><td rowspan="2">Emphysema
“Pink Puffers”</td><td>Definition:
It is a pathological diagnosis: permanent enlargement of air spaces distal to terminal bronchioles due to destruction of alveolar walls, without obvious fibrosis.</td></tr>
<tr><td>Pathogenesis:
Destruction of alveolar walls due to an increase in protease or a decrease in α1-antitrypsin.
This destruction reduces the elastic recoil and the airways collapse during expiration.</td></tr>
<tr><td>Risk factors</td><td colspan="2">1. Tobacco smoke
2. α-1 antitrypsin deficiency
3. environmental (second hand smoking)
4. chronic asthma
5. recurrent infections
6. pollution (biomass fuel)
7. occupational exposure (e.g. coal, silica)</td></tr>
</table>

<table>
<tr><td>Clinical presentation</td><td colspan="2">❖ cough with white or clear sputum (purulent in infective exacerbation).
❖ wheeze and breathlessness.
❖ tachypnea, tachycardia.
❖ cyanosis.
❖ prolonged expiratory time.
❖ Use of accessory respiratory muscles.
❖ Signs of cor pulmonale.
❖ Edema and morning headaches.</td><td>1. Auscultation:
● End expiratory wheeze
● Decreased breath sounds
● +/- inspiratory crackles
2. Percussion:
● Hyperresonance</td></tr>
<tr><td>Investigations</td><td colspan="3">1. (definitive diagnostic test): Lung function test (Spirometry) and apply GOLD criteria. (decreased FEV1 & FEV1/FVC ratio)
2. Chest X-ray (low sensitivity for diagnosis but useful in acute exacerbation)
3. High resolution CT scan.
4. Measure α1-antitrypsin levels
5. Arterial blood gas (ABG)</td></tr>
<tr><td rowspan="2">Management</td><td colspan="3">Improves mortality:
➢ Smoking cessation
➢ Long term oxygen therapy
➢ vaccinations</td></tr>
<tr><td colspan="3">Improve symptoms:
➢ Bronchodilators
➢ Corticosteroids
➢ Pulmonary rehabilitation
➢ Surgery</td></tr>
</table>

Bronchiectasis Summary

Definition	Abnormal permanent dilation and irreversible damage of bronchi.
Pathogenesis	There is permanent, abnormal dilation and destruction of bronchial walls with chronic inflammation, airway collapse, and ciliary loss/dysfunction leading to impaired clearance of secretion.
Causes	• Cystic fibrosis • Hypogammaglobulinemia • Ciliary dysfunction (immotile cilia syndrome, Kartagener's syndrome) • Foreign body or tumors (cause obstruction) • Infections (aspergillosis, H. influenzae, measles, mycobacterium, Staph. Aureus, P. aeruginosa).
Clinical features	❖ Chronic cough with large amounts of mucopurulent foul-smelling sputum. ❖ Dyspnea, weight loss, fever ❖ Hemoptysis ❖ Recurrent or persistent pneumonia ❖ Wheezes or crackles
Investigations	1. High resolution CT (HRCT) (most accurate test, study of choice) 2. Chest X-ray (CXR) best initial test (can show abnormalities in advanced cases) 3. Sputum culture 4. PFT 5. screening for diseases that cause this condition. 6. Bronchoscopy (in certain cases).
Management	• Bronchodilators and corticosteroids • Bronchial hygiene: ➢ hydration ➢ Chest physiotherapy (helps to remove the mucus) • Antibiotics (for acute exacerbations) • Surgical (in few cases)

Examine Yourself !!

1) Which of the following are the most likely physical examination findings in a patient with emphysema?

A. Diffuse expiratory wheezing
B. Clubbing of the fingers
C. Bibasilar inspiratory crackles with increased jugular venous pressure (JVP)
D. Inspiratory stridor
E. Third heart sound

2) A 56-year-old woman admits to a 60-pack-year smoking history. She complains of fatigue and dyspnea with minimal exertion, and a cough that is productive each morning. Which of the following is the most likely finding in this patient?

A. Normal diffusing capacity of lung for carbon monoxide (DLCO).
B. Decreased residual volume.
C. Normaltoslightlyincreasedforcedexpiratoryvolumeinfirstsecond (FEV1).
D. Decreased forced expiratory volume in first second/forced vital capacity (FEV1/FVC).
E. Decreased forced vital capacity(FVC)

3) 40 years old lady came complaining of 2 years shortness of breath and cough in the pulmonary clinic. She admitted that she was a smoker for 20 years, has a cat and work in chocolate factory. PFT done and obstructive pattern was found, a trial of bronchodilators were not effective. What is the most likely diagnosis?

A. COPD
B. Kyphoscoliosis
C. Asthma
D. Hypersensitivity pneumonitis

4) Which of the following is true regarding the etiology of bronchiectasis?

A. Asthma is well known cause of bronchiectasis.
B. Dry bronchiectasis indicates upper lobe bronchiectasis and might be caused by TB
C. Childhood infection is not a risk factor.
D. Exposure to dust and air pollution is a known risk factor.

5) Which of the following therapies is most likely to provide the greatest benefit to a patient with chronic stable emphysema and a resting oxygen saturation of 86%?

A. Inhaled tiotropium daily
B. Inhaled albuterol as needed
C. Oral prednisone daily
D. Supplemental oxygen used at night
E. Supplemental oxygen used continuously

6) You see a 68-year-old man in clinic, with a 40 (cigarette) pack year history, who has been experiencing breathlessness on exertion and a productive cough of white sputum over the last four months. You assess his spirometry results which reveal an FEV1/FVC of 51 per cent with minimal reversibility after a 2-week trial of oral steroids. Cardiological investigations are normal. Which of the following is the most likely diagnosis?

A. Asthma
B. Chronic obstructive pulmonary disease (COPD)
C. Left ventricular failure
D. Chronic bronchitis
E. Lung fibrosis

7) The severity of COPD is assessed using post bronchodilator spirometry analysis. From the list below, select the values that you would expect to see in a patient with moderate COPD.

A. FEV1/FVC <0.7, FEV1 per cent predicted 30–49 %
B. FEV1/FVC <0.7, FEV1 per cent predicted ≥80 %
C. FEV1/FVC <0.7, FEV1 per cent predicted <30 %
D. FEV1/FVC <0.7, FEV1 per cent predicted 50–79 %
E. FEV1/FVC <0.7, FEV1 per cent predicted 60–70 %

Answers:

1) A . COPD is characterized by chronic airway obstruction, with most airflow resistance occurring in small airways of the lower respiratory tract, producing expiratory wheezing. Inspiratory stridor would occur with upper airway, usually extrathoracic, obstruction. Clubbing is not generally a feature of COPD and should prompt investigation for another disease process such as a bronchogenic carcinoma. Crackles, elevated JVP, and an S3 are signs of congestive heart failure.

2) D . This patient likely has COPD, based on the smoking history and symptoms. A decrease in the forced expiratory volume in first second/ forced vital capacity ratio is the hallmark of airflow obstruction. The FEV1 is decreased in obstructive, as well as in restrictive, lung disease. The diffusing capacity is typically deceased in COPD as well as intrinsic restrictive lung disease. The DLCO indicates the adequacy of the alveolar-capillary membrane; the residual volume is the volume of air remaining in the lungs after a maximal expiratory effort and is usually increased in COPD dueto air trapping.

3) A , 4) B

5) E. For patients with chronic hypoxemia, supplemental oxygen has a significant impact on mortality, with a greater benefit with continuous usage, rather than intermittent or nocturnal-only usage. Bronchodilators such as tiotropium and albuterol improve symptoms and improve FEV1, but offer no mortality benefit. Chronic use of oral corticosteroids should be avoided because of unfavorable side effects such as osteoporosis, glucose intolerance, and gastrointestinal (GI) side effects.

6) B. The patient's symptom history coupled with the spirometry results indicate that he has an obstructive defect. Spirometry is typically used to measure functional lung volumes. The ratio of the forced expiratory volume in one second (FEV1) to the forced vital capacity (FVC), provides a reliable approximation of severity of airflow obstruction; the normal being 80 per cent. An FEV1/FVC ratio of less than 80 per cent indicates an obstructive defect seen in COPD and asthma while a ratio of greater than 80 per cent is representative of a restrictive defect seen in lung fibrosis. (E). The spirometry results coupled with minimal reversibility points the diagnosis to COPD (B) rather than asthma (A), where reversibility of the FEV1/FVC ratio is usually seen. Chronic bronchitis (D) can be defined as cough productive of sputum for three months of two successive years which does not corroborate with the onset of symptoms. Left ventricular failure (C) is obviously incorrect due to the fact that cardiological tests have been mentioned as normal.

7) D. With reference to the NICE guidelines 2010, COPD can be divided into mild, moderate, severe and very severe. The values are obtained with post bronchodilator spirometry and are as follow: Mild COPD: FEV1/FVC <0.7, FEV1 % predicted ≥80 per cent (B) Moderate COPD: FEV1/FVC <0.7, FEV1 % predicted 50–79 per cent (D) Severe COPD: FEV1/FVC <0.7, FEV1 % predicted 30–49 per cent (A) Very severe COPD: FEV1/FVC <0.7, FEV1 % predicted <30 per cent (C) Very severe COPD can also be seen in patients with FEV1 % predicted <50 per cent with respiratory failure.