CHRONIC OBSTRUCTIVE PULMONARY DISEASE
DEFINITION OF COPD

- **Chronic obstructive pulmonary disease** (COPD) is a preventable and treatable disease state characterized by air flow limitation that is not fully reversible.

- **Air flow limitation** is usually progressive and is associated with an abnormal inflammatory response of lungs to noxious particles or gases, primarily caused by cigarette smoking.
Component of COPD

- The definition *Include* chronic bronchitis, emphysema with airflow limitation.

- The definition *exclude* other causes of chronic airflow obstruction such as Pulmonary cystic fibrosis, diffuse panbronchiolitis and bronchiecstasis etc.
Process of copd

- Chronic Bronchitis
- Obstructive emphysema
- COPD (airflow limitation)
- Pulmonary artery hypertension
- Col pulmonal heart disease
Chronic Bronchitis
Chronic Bronchitis

- *Chronic bronchitis* is defined clinically as the presence of a cough productive of sputum not attributable to other causes on most days for at least 3 months over 2 consecutive years.

- Clinical and epidemiological term
Chronic Bronchitis

- Chronic nonspecific inflammation
- Symptoms of cough and sputum production with or without gasping
- Recurrent attacks
- Chronic proceeding
## Classification of Chronic Bronchitis

<table>
<thead>
<tr>
<th>Simple type of Chronic Bronchitis (without gasping)</th>
<th>Cough</th>
<th>Sputum expectoration</th>
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<tr>
<td>Chronic Bronchitis with gasping</td>
<td>Cough</td>
<td>Sputum expectoration</td>
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<tr>
<td></td>
<td></td>
<td><strong>Gasping</strong></td>
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</tbody>
</table>
## Stages of Chronic Bronchitis

<table>
<thead>
<tr>
<th>Stages</th>
<th>Time Courses</th>
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<tbody>
<tr>
<td>Exacerbation</td>
<td>In a week</td>
</tr>
<tr>
<td>Chronic lag phase</td>
<td>One month or longer</td>
</tr>
<tr>
<td>stable</td>
<td>Lasts for two months</td>
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</tbody>
</table>
Diagnosis of chronic bronchitis

- Cough & Sputum expectoration & Gasping
- Three months /per year or longer
- Continuously longer than two years
- Exclude other lung and heart disease

If shorter than three months /per year then definite objective evidences are demanded (such as X-Ray and lung function et al.) to diagnose.
Obstructive Emphysema
Definition of Emphysema

Pulmonary emphysema
(a pathological term)
is characterized by abnormal, permanent enlargement of air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and hyperdistension leading to reduction in lung elastics recoil and airway obstruction.
## Classification of Emphysema

<table>
<thead>
<tr>
<th>Obstructive Emphysema</th>
<th>Senile emphysema (Physiological)</th>
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<tr>
<td>Emphysema without Obstruction</td>
<td>Interstitial Emphysema</td>
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<tr>
<td></td>
<td>Compensating Emphysema</td>
</tr>
<tr>
<td></td>
<td>Scarred Emphysema</td>
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</tbody>
</table>

- **Obstructive Emphysema**
  - Senile emphysema (Physiological)
  - Interstitial Emphysema
  - Compensating Emphysema
  - Scarred Emphysema
Risk factor for COPD

Genes
Exposure to particles
• Tobacco smoke
• Occupational dusts, organic and inorganic
• Indoor air pollution from heating and cooking with biomass in poorly vented dwellings
• Outdoor air pollution

Lung Growth and Development
Oxidative stress
Gender/ Age/ Respiratory infections /Socioeconomic status
Nutrition
Comorbidities
Pathogenesis of COPD

Cigarette smoke
Biomass particles and particulates

Host factors and amplifying mechanisms

Anti-oxidants

Oxidative stress
Proteinases

Anti-proteinases

Repair mechanisms

Lung inflammation

COPD pathology
Pathogenesis

The inflammation in the respiratory tract of COPD patients appears to be an amplification of the normal inflammatory response of the respiratory tract to chronic irritants such as cigarette smoke.

**Inflammatory Cells**

Involve neutrophils, macrophages, and lymphocytes. These cells release inflammatory mediators and interact with structural cells in the airways and lung parenchyma.

**Inflammatory Mediators**

The inflammatory mediators attract inflammatory cells from the circulation (chemotactic factors), amplify the inflammatory process (proinflammatory cytokines), and induce structural changes (growth factors).
Oxidative Stress

Oxidative stress may be an important amplifying mechanism in COPD. Oxidants are generated by cigarette smoke and other inhaled particulates, and released from activated inflammatory cells such as macrophages and neutrophils.

Protease-Antiprotease Imbalance

Protease-mediated destruction of elastin, a major connective tissue component in lung parenchyma, is an important feature of emphysema and is likely to be irreversible.
Although both COPD and asthma are associated with chronic inflammation of the respiratory tract, there are marked differences in the inflammatory cells and mediators involved in the two diseases, which in turn account for differences in physiological effects, symptoms, and response to therapy.
Pathological changes characteristic of COPD are found in
the proximal airways,
peripheral airways,
lung parenchyma,
and pulmonary vasculature.

These changes include chronic inflammation, and
structural changes.
• **Proximal airways (trachea, bronchi > 2 mm internal diameter)**

  - Goblet cells, enlarged submucosal glands (both leading to mucus hypersecretion), squamous metaplasia of epithelium

• **Peripheral airways (bronchioles < 2mm i.d.)**

  - Airway wall thickening, peribronchial fibrosis, luminal inflammatory exudate, airway narrowing (obstructive bronchiolitis)

  - Increased inflammatory response and exudate correlated with disease severity.
• Lung parenchyma (respiratory bronchioles and alveoli)

Alveolar wall destruction, apoptosis of epithelial and endothelial cells.

• **Centrilobular emphysema**: dilatation and destruction of respiratory bronchioles; most commonly seen in smokers

• **Panacinar emphysema**: destruction of alveolar sacs as well as respiratory bronchioles; most commonly seen in alpha-1 antitrypsin deficiency
Normal distal lung acinus
Centriacinar (centrilobular) emphysema
Panacinar emphysema
• Pulmonary vasculature
  Thickening of intima, endothelial cell dysfunction, → smooth muscle → pulmonary hypertension.
Airflow Limitation and Air Trapping

- The inflammation, fibrosis, and luminal exudates in small airways is correlated with the reduction in FEV1 and FEV1/FVC ratio.
- The peripheral airway obstruction traps air during expiration, resulting in hyperinflation.
- Emphysema is more associated with gas exchange abnormalities than with reduced FEV1.
Gas Exchange Abnormalities

VA/Q imbalance

Reduced pulmonary vascular bed

Mucus Hypersecretion

Pulmonary Hypertension

• Hypoxic vasoconstriction of small pulmonary arteries eventually result in structural changes that include intimal hyperplasia and later smooth muscle hypertrophy/hyperplasia.

• The loss of the pulmonary capillary bed in emphysema may also contribute to increased pressure in the pulmonary circulation.

• Progressive pulmonary hypertension may lead to right ventricular hypertrophy and eventually to right-side cardiac failure (cor pulmonale).
**Systemic features**

They have a major impact on survival and comorbid diseases.

- Cachexia
  - a loss of skeletal muscle mass and weakness
- increased likeliness of having osteoporosis, depression and chronic anemia.
- Increased concentrations of inflammatory mediators, including TNF-α, IL-6, and oxygen-derived free radicals,
- There is an increase in the risk of cardiovascular diseases.
Clinical Manifestation

History:
- History of exposure to risk factors, Tobacco smoke.
- Occupational dusts and chemicals
- Smoke from home cooking and heating fuels.
- Age of onset: After middle age
- Season: winter
Clinical Manifestation

Symptoms:

- Gradually progressive dyspnea is the most common presenting character.

Dyspnea that is:
- Progressive (worsens over time)
- Usually worse with exercise
- Persistent (present every day)
- Described by the patient as an “increased effort to breathe,” “heaviness,” “air hunger,” or “gasping.”
• Chronic Cough
  May be intermittent and may be unproductive.
• Chronic sputum production:
  • Recurrent respiratory infection
  • Recurrent attacks leading to cor pulmonal heart disease
  • Unexpected weigh loss
  • Decreased food appetite
Physical Signs:

- **Earlier period:** Minimal/Nonspecific signs
- **Advanced Stage:**
  *Inspection:* 
  Barrel-shaped chest, accessory respiratory muscle participate, prolonged expiration during quiet breathing.
  
  *Palpation:* 
  Weakened fremitus vocalis
Clinical Manifestation

*Percussion:
Hyperresonant depressed diaphragm, diminution of the area of absolute cardiac dullness.

*Auscultation:
Prolonged expiration; reduced breath sounds;
The presence of wheezing during quiet breathing
Crackle can be heard if infection exist.
The heart sounds are best heard over the xiphoid area.
Auxillary Examination

Chronic bronchitis

- Chest Radiograph (X-Ray)
  Non apparent abnormality
  Or thickened and increased of the lung markings are noted.
Auxiliary Examination

Chest X-Ray -- emphysema

- Chest findings are also variable.
- Marked over inflation is noted with flattend and low diaphragm
- Intercostal space becomes widen
- A horizontal pattern of ribs
- A long thin heart shadow
- Decreased markings of lung peripheral vessels
Chest X-Ray
Auxiliary Examination

Pulmonary function Test

- Determination of a forced vital capacity and FEV1 is necessary for the diagnosis and assessment of the severity of the disease and helpful in following its progress.

- FEV1/FVC is the best index of airflow obstruction.
Auxiliary Examination

Pulmonary function Test

diagnostic criteria

A post-bronchodilator

- \((\text{FEV1})/\text{forced vital capacity (FVC)} \leq 70\%\)
  confirm the presence of airflow limitation that is not fully reversible.

- \(\text{FEV1} \%\text{pred}\) is used for evaluation of the severity of pulmonary function status.

The FEV1 and the FEV1/FVC ratio fall progressively as the severity of COPD increases.
Pulmonary function Test

- Elevations of total lung capacity (TLC)
- Functional reserve capacity (FRC)
- Residual volume (RV)
- RV/TLC > 40% for emphysema
- Vital capacity (VC)
- Peak expiratory flow (PEF)
### Spirometric Classification of COPD

<table>
<thead>
<tr>
<th>Category</th>
<th>FEV1/FVC</th>
<th>FEV1%pred</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt;70%</td>
<td>≥80%</td>
</tr>
<tr>
<td>Moderate</td>
<td>&lt;70%</td>
<td>50~80%</td>
</tr>
<tr>
<td>Severe Disease</td>
<td>&lt;70%</td>
<td>30~50%</td>
</tr>
<tr>
<td>Very Severe</td>
<td>&lt; 70%</td>
<td>≤ 30% or &lt;50%</td>
</tr>
</tbody>
</table>

Following with respiratory failure & right heart failure.
Auxiliary Examination

CT (Computed tomography): greater sensitivity and specificity for emphysema than CXR, especially for the diagnosis of bronchiectasis and evaluation of bullous disease.
Computed Tomography
**Laboratory Examination**

- **Blood examination**
  In exacerbation or acute infection in airway, leucocytosis may be detected.

- **Sputum examination**
  - Streptococcus pneumonia
  - Haemophilus influenzae
  - Moraxella catarrhalis
  - Klebsiella pneumonia
Blood gas analysis:

Arterial blood gas analysis may reveal hypoxemia, particularly advanced disease.

In patients with severe hypoxemia, CO2 retention, it shows low arterial PO2 and high arterial PCO2.
Diagnosis of COPD

- Clinical manifestation
- Auxiliary examinations
- Significant importance of Pulmonary function test

Spirometry should be obtained in all patients with:
- Exposure to cigarettes;
  Environmental or occupational pollutants;
- presence of cough, sputum production or dyspnea
Stage:

- **Exacerbation:**
  Gradually progressive Cough and sputum & Dyspnea and gasping
  Increased purulence sputum followed by recurrent respiratory infection.

- **Stable:**
  Stable systems of Cough and sputum, gasping and dyspnea are alleviated.
Differential Diagnosis of COPD

**Diagnosis**
- **COPD**
  1. Mid-life onset
  2. Slowly progressing symptoms
  3. Long history of smoking
  4. Dyspnea during exercise
  5. largely irreversible airflow limitation

- **Asthma**
  1. Early onset
  2. Symptoms vary from day to day
  3. Symptoms at the night/early morning
  4. A family history
  5. Airflow limitation that is largely reversible
  6. largely reversible airflow limitation
  7. Allergy, rhinitis, eczema
Differential Diagnosis of COPD

**Diagnosis**

- **Pulmonary carcinoma**
  - Commonly occurs in patients over 40 years old
  - with cigarette smoking.
  - Obvious radiological abnormality

- **Tuberculosis**
  - Onset at all ages
  - Tuberculosis toxic syndrome
  - Lung infiltrate on chest radiography
  - Microbiological confirmation

  Sputum examination of positive TB bacterium can confirm the diagnosis
Differential Diagnosis of COPD

Diagnosis

- Bronchiectasis
- Non-obstructive emphysema

Suggestive features

1. Large volume of purulent sputum
2. Commonly associated with bacterial infection
3. Coarse crack/clubbing on auscultation
4. Bronchial dilation and bronchial wall thickening on X-ray/CT

pulmonary function tests
Complication

- chronic respiratory failure
- Pneumothorax
- Chronic pulmonary heart disease
TREATMENT

Aim

Based on the principles of

- prevention of further progress of disease
- preservation and enhancement of pulmonary functional capacity
- avoidance of exacerbations in order to improve the quality of life.
TREATMENT

- Stop smoking
- Avoid environment pollution
- Antibiotic therapy
- Bronchodilators
- Glucocorticoids
- Expectorant
- Respiratory stimulant
- Oxygen therapy
- Rehabilitation care
- Lung volume reduction surgery
stable COPD(I):

avoid risk factors

- Education and smoking cessation
  
  *Smoking cessation has the greatest capacity to influence the natural history of COPD.*

- Control the occupational and environmental pollution
Drug therapy:

- Prevent and control symptoms,
- Increase exercise capacity,
- Reduce the frequency and severity of exacerbations,
- Improve health status.
1. Bronchodilators—

Bronchodilators are central to the symptomatic management of COPD.

- improve emptying of the lungs, reduce dynamic hyperinflation and improve exercise performance.
Drug Therapy

Bronchodilators

Three major classes of bronchodilators:

- **β2 - agonists:**
  - Short acting: salbutamol & terbutaline
  - Long acting: Salmeterol & formoterol

- **Anticholinergic agents:**
  - Ipratropium, tiotropium

- **Theophylline** (a weak bronchodilator, which may have some anti-inflammatory properties)
2. Glucocorticoids

- Regular treatment with inhaled glucocorticoids is appropriate for symptomatic patients with an FEV1 < 50% pred and repeated exacerbations.
- Chronic treatment with systemic glucocorticoids should be avoided because of an unfavorable benefit-to-risk ratio.
3. COMBINATION THERAPY

Combination therapy of long acting β2-agonists and inhaled corticosteroids show a significant additional effect on pulmonary function and a reduction in symptoms.

*Mainly in patients with an FEV1<50%pred*
Drug Therapy

4. Others:
- Antioxidant agents
- Immunoregulators
- Vaccine
- Alpha-1 antitrypsin augmentation
- Mucolytic (mucokinetic, mucoregulator) agents
- Antitussives
Oxygen Therapy

- **Oxygen** -- >15 h /d

Long-term oxygen therapy (LTOT) improves survival, exercise, sleep and cognitive performance in patients with respiratory failure.

The therapeutic goal is to maintain SaO2 ≥ 90% and PaO2 ≥ 60mmHg at sea level and rest.
Long-term Oxygen therapy
LTOT

Indication:

- For patients with a
  \( \text{PaO}_2 \leq 55 \text{ mmHg or SaO}_2 \leq 88\% \),
  with or without hypercapnia

- For patients with a
  \( \text{PaO}_2 \text{ of 55~70} \ (60) \text{ mmHg or SaO}_2 \leq 89\% \)
  as well as pulmonary hypertension / heart failure / polycythemia
  (hematocrit >55\%)
- **Pulmonary rehabilitation**
- **Nutrition**
- **Surgery:**
  - Bullectomy
  - Lung volume reduction surgery
  - Lung transplantation
Manage exacerbation

- **Identify the cause of exacerbation:**
  Virus or Bacteria or Other uncertain reasons

- **Assessment of severity:**
  The proceeding history and disease must be considered and comparison is very important.
**Oxygen therapy**

**Controlled oxygen therapy.**

Supplemental oxygen should be titrated to improve the patient’s hypoxemia. Adequate levels of oxygenation (PaO2 > 8.0 kPa, 60 mm Hg, or SaO2 > 90%) are easy to achieve in uncomplicated exacerbations, but CO2 retention can occur insidiously with little change in symptoms. Once oxygen is started, arterial blood gases should be checked 30-60 minutes later to ensure satisfactory oxygenation without CO2 retention or acidosis.
**Bronchodilators:**
Increase dose and times properly
Atomization and inhalation

**Glucocorticoids:**
Oral or intravenous glucocorticosteroids are recommended. Thirty to 40 mg of oral prednisolone daily for 7-10 days is effective and safe.
**Antibiotics**

Respiratory infection is the usual predisposing factor.

It is advocated to select antibiotics according to culture of sputum and drug-sensitivity test.

**Mechanical Ventilation**

- Noninvasive mechanical ventilation
- Invasive mechanical ventilation

**Others:**
THANK YOU