Chronic Obstructive Pulmonary Disease

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COPD is a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema, the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity, and that is not fully reversible. In developed countries, cigarette smoking accounts for over 90% of cases. However, only 10—20 % of heavy smokers develop COPD, indicating individual susceptibility.

It is unusual to develop COPD with less than 10 pack years .

Number of pack-years = (number of cigarettes smoked per day/20) × number of years smoked.

Prevalence rates for COPD are correlated with increasing age, lower socioeconomic status, and smoking.

Cigarette smoking is by far the most common cause of COPD; however, other factors such as inhalation of cooking fire smoke, air pollution, occupational exposures to dust and fumes, and infections contribute to the occurrence, severity, and progression of the disease.

Chronic Bronchitis:

Persistent cough resulting in sputum production for more than 3 months in each of the past 2 years. Cigarette smoking is the major cause, although exposure to pollutants such as dusts may also play a role.

pathological finding is hypertrophy and increase in number of the mucus-secreting goblet cells of the bronchial tree. Microscopically there is infiltration of the walls of the bronchi and bronchioles with acute and chronic inflammatory cells and lymphoid follicles in severe disease. In contrast to asthma, the lymphocytic infiltrate is predominantly CD8+. The epithelial layer may become ulcerated and‹ when the ulcers heal, squamous epithelium may replace the columnar cells. The inflammation is followed by scarring and a remodeling process that thickens the walls and leads to widespread narrowing in the small airways.

Emphysema:

is defined pathologically as dilatation and destruction of the lung tissue distal to the terminal bronchiole. It is classified according to the site of damage:

Centri-acinar emphysema.

Damage around the respiratory bronchioles, whilst the more distal alveolar ducts and alveoli tend to be well preserved. This form of emphysema is extremely common.

Pan-acinar emphysem:

This is less common. Distension and destruction appear to involve the whole of the acinus, and in the extreme form the lung becomes a mass of bullae. Severe airflow limitation and *V/Q* mismatch occur. This type of emphysema occurs in α1-antitrypsin deficiency.

The only genetic disorder thus far definitively linked to COPD is α1-antitrypsin deficiency, which accounts for less than 1% of all cases. A small minority develop liver disease.The deficient enzyme, α1-antitrypsin, an acute-phase reactant, is produced primarily in the liver, from which it travels to the lung, where it deactivates elastases released by inflammatory cells that are capable of degrading connective tissue matrices.

Clinical features:

cough with the production of sputum, wheeze and slowly progressive dyspnea following many years of a smoker's cough. and frequent infective exacerbations occur, giving purulent sputum. Symptoms can be worsened by factors such as cold, foggy weather and atmospheric pollution. With advanced disease, breathlessness becomes severe even after mild exercise such as dressing affected individuals complain of exercise intolerance and fatigue, and the disease eventually leads to weight loss, depression, and anxiety as a result of increased work of breathing.

physical examination:

During the early stages of COPD, the physical examination may be normal. A normal examination and the absence of symptoms often delay diagnosis.so Inspection of the thorax and palpation may fail to reveal findings. As the disease progresses, patient the appearance of "barrel chest."

The lungs may be hyper resonant to percussion.

Auscultation may show diminished breath sounds with rhonchi, wheezes, or faint crackles.

During the late stages of COPD, patients show evidence of increased work of breathing with use of accessory muscles, pursed-lip breathing, and weight loss. Skeletal muscle wasting may also become evident.

Despite their respiratory insufficiency, some patients are able to sustain relatively normal oxygen levels in blood until very late in the disease, leading to the classic clinical presentation of the **"pink puffer."** This is in emphysema

Other patients tend to retain CO2 and diminish their work of breathing, resulting in chronic respiratory acidosis and, in extreme cases, polycythemia and cyanosis; and Called **"blue bloater**". This is in Chronic Bronchitis

As the disease progresses, the lung volumes increase (hyperinflation) and the diaphragms flatten, which renders inspiratory excursions inefficient.

In advanced disease, the cardiovascular system becomes affected as a result of loss of vasculature in destroyed alveolar walls and vascular remodeling due to chronic hypoxia. With limited area for blood flow, pulmonary vascular resistance is increased, leading to increased right ventricular afterload and development of pulmonary hypertension. This accelerates the development of right ventricular failure, which is referred to as **cor pulmonale** in the setting of lung disease.

Right heart gallop, loud pulmonary second sound may be heard.distended neck veins, hepatojugular reflux, may develop with a greatly elevated jugular venouspressure (JVP), ascites and upper abdominal discomfort due to swelling of the liver. and leg edema characterize cor pulmonale .Commonly associated comorbid conditions include cardiovascular disease, cerebrovascular disease, the metabolic syndrome, osteoporosis, depression and lung cancer.

Investigation:

Pulmonary function tests

Measuring peak expiratory flow(PEFR) is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

Spirometry

Decreased FEV1.

Reduction of the FEV1/FVC ratio .

The severity of COPD is categorised using the FEV1:
Post-bronchodilator

-FEV1/FVC	FEV1(of predicted)	Severity
< 0.7	> 80%	Stage 1 – Mild
< 0.7	*50-79%*	Stage 2 Moderate
< 0.7	30-49%	Stage 3 -Severe
< 0.7	< 30%	Stage 4 -Very severe

Chest X-ray:

is often normal, even when the disease is advanced. The classic features are the presence of bullae ,severe overinflation of the lungs with low, flattened diaphragms,

Haemoglobin level and PCV: can be elevated as a result of persistent hypoxaemia (secondary poly cythaemia).

Blood gases are often normal. In the advanced case there is evidence of hypoxaemia and hypercapnia.

Sputum examination is unnecessary in the ordinary case . Strep, pneumoniae or H. influenzae are the only common organisms to produce acute exacerbations. Occasionally Moraxella catarrhalis may cause infective exacerbations.

Electrocardiogram. In advanced cor pulmonale the P wave is taller (P pulmonale) and there may be right bundle branch block.

Echocardiogram :performed to assess cardiac function.

***α* -Antitrypsin levels.** The normal range is 2-4 g/L.

COPD Management:

General management:

Smoking cessation advice will decrease mortality in patients with COPD who do succeed at quitting. stop smoking is vital. Even at a late stage of the disease this may slow down the rate of deterioration and prolong the time before disability.

Annual influenza vaccination.

pneumococcal vaccination. single dose usually provides lifelong immunity.

the short -term management of exacerbations and for the long-term relief of symptoms. In many cases the drugs used are similar to those used in asthma.

Bronchodilator therapy:

A short-acting beta2-agonist (SABA) Salbutamole , Albuterol or

Short-acting muscarinic antagonist (SAMA) Ipratropium bromide. Is first-line treatment even can use in combination. In practice, a combination of albuterol and ipratropium is frequently prescribed because these agents produce greater benefits when used in combination than individually.

For patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by the FEV1:

FEV1 > 50% use:

Long-acting beta2-agonist (LABA), salmeterol, or
Long-acting muscarinic antagonist (LAMA), tiotropium.

FEV1 < 50%:

→LABA + **inhaled corticosteroid** (ICS) in a combination inhaler.

For patients with persistent exacerbations or breathlessness ,the chronic use of inhaled corticosteroids improves symptoms and decreases the frequency of exacerbations.

For this reason, inhaled long-acting corticosteroids (e.g., budesonide) are frequently used in the treatment of COPD

LABA + inhaled corticosteroid (ICS) in a combination inhaler.

Antibiotics:

Prompt antibiotic treatment shortens exacerbations and should always be given in acute episodes as it may prevent hospital admission and further lung damage. Antibiotics for patients reporting an increase in sputum purulence, sputum volume or breathlessness .Most cases simple regimens are advised, such as A co-amoxiclav, tetracycline or a macrolide.

Oral theophylline, only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy, the dose should be reduced if macrolide or fluoroquinolone antibiotics are prescribed.

Mucolytics, Should be 'considered' in patients with a chronic productive cough and continued if symptoms improve Cor pulmonale features .

Oxygen therapy

Long-term domiciliary oxygen therapy (LTOT) improves survival in selected patients with COPD complicated by severe hypoxaemia (arterial PaO_2 < 7.3 kPa (55 mmHg);

Phosphodiesterase-4 Inhibitors.

The most recent addition to pharmacotherapy for COPD, roflumilast, has shown efficacy and tolerability in patients with severe COPD.

Roflumilast is not indicated for the relief of acute bronchospasm or rescue therapy. It is an expensive medication and is used primarily as add-on therapy in patients with severe disease not adequately controlled on other COPD medications.

Common adverse events include diarrhea, weight loss, nausea, and headache. Psychiatric adverse events.

Oral glucocorticoids: are useful during exacerbations but not for maintenance therapy due to side effect as osteoporosis and impaired skeletal muscle function, and should be avoided.

Pulmonary rehabilitation: (consisting of education, nutritional counseling, exercise training, assessment, and follow-up).

Surgical intervention : Large bullae (bullectomy). Lung volume reduction surgery .

Lung transplantation may benefit carefully selected patients with advanced disease.

Other Agents:

α1-Antitrypsin replacement therapy. Weekly or monthly infusions of α1-antitrypsin have been recommended for patients with serum levels of this compound below 310 mg/L and abnormal lung function.

Antioxidant agents (particularly N-acetylcysteine).

Nedocromil and leukotriene modifiers have not been adequately tested in COPD.

Routine use of antitussives is not recommended in patients with stable COPD because cough has a significant protective role.

Factors which may improve survival in patients with stable COPD:

Smoking cessation - the single most important intervention in patients who are still smoking

Long term oxygen therapy(**LTOT**)in patients who fit criteria:

Arterial blood gases measured in clinically stable patients on optimal medical therapy on at least two occasions 3 weeks apart:

PaO2 < 7.3 kPa (55 mmHg) irrespective of PaCO2 and FEV1 < 1.5 L

PaO2 7.3–8 kPa (55–60 mmHg) plus pulmonary hypertension, peripheral oedema or nocturnal hypoxaemia

The patient has stopped smoking.

Use at least 15 hrs/day at 2–4 L/min to achieve a PaO2 > 8 kPa (60 mmHg) without unacceptable rise in PaCO2

LTOT should be offered to patients with :

ApO2 of < 7.3 kPa or to those with

A pO2 of 7.3 - 8 kPa and one of the following:

Secondary polycythaemia.

Nocturnal hypoxaemia.

Peripheral oedema.

Pulmonary hypertension.

Acute exacerbations of COPD:

Acute exacerbations of COPD are characterised by an increase in symptoms and deterioration in lung function and health status.

Usually triggered by bacteria . The most common bacterial pathogens in COPD are(Streptococcus pneumoniae, Haemophilus inflenzae, and Moraxella catarrhalis.) ,viruses or a change in air quality.

They may be accompanied by the development of respiratory failure and/ or fluid retention, cough, sputum production and are an important cause of death.

Many patients can be managed at home with the use of increased bronchodilator therapy, a short course of oral corticosteroids and, if appropriate, antibiotics. The presence of cyanosis, peripheral oedema or an alteration in consciousness indicates the need for referral to hospital.

Management Acute exacerbations of COPD:

Controlled oxygen at 24% or 28% should be used with the aim of maintaining PaO2 above 8 kPa (60 mmHg) (or an SaO2 between 88% and 92%) without worsening acidosis.

Nebulised short-acting β2-agonists, combined with an anticholinergic agent (e.g. s**albutamol and ipratropium**). With careful supervision, it is usually safe to drive nebulizers with oxygen .

Intravenous corticosteroids then transitioned from intravenous to oral steroids within 72 hours, with a subsequent tapering of the oral steroid dose over 2 weeks (Oral prednisolone)

Antibiotics for patients reporting an increase in sputum purulence, sputum volume or breathlessness Antibiotic therapy is most beneficial in treating infectious exacerbations of COPD that are characterized by increases in **dyspnea**, **sputum** volume, and sputum **purulence**. Antibiotic therapy also is indicated in patients with severe exacerbations of COPD who require

mechanical ventilation, with previous antibiotics mentions above , A fluoroquinolone such as levofloxacin or the combination of a third-generation cephalosporin (cefixime)plus a macrolide antibiotic(azithromycin) will usually cover the most common bacterial pathogens (Hemophilus influenzae, Streptococcus pneumoniae, and Moraxella catarrhalis as well as causes of “atypical” pneumonia).
Diuretics (when development of peripheral edema).