CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

BACKGROUND

COPD is a cause of significant suffering and is the third leading cause of death in the USA.[1] COPD includes chronic bronchitis and emphysema. COPD is a slowly progressive disease involving the airways and/or pulmonary parenchyma leading to airflow obstruction. Inflammatory cells (T lymphocytes, neutrophils, and alveolar macrophages) release enzymes that attack bronchial tissue. In chronic bronchitis airways become chronically inflamed with mucus production. In emphysema alveoli are damaged and eventually destroyed resulting in the development of bullae.

Tobacco smoking is the most common risk factor for COPD. Smokers have poorer outcomes than non-smokers. Other risk factors include air pollution, second hand smoke, and history of childhood respiratory infections. Urban areas with heavy traffic, poor housing, poor sanitation, garbage accumulation, and poor drainage and sewage systems have high rates of COPD. Other risk factors include industrial pollutants such as grains, heavy metals, welding fumes, and coal.

In some cases genetics play a role. The ADAM33 gene is more common in smokers with COPD than in those who do not have the disease. Genetic variants such as, disorders of chromosome 4, and a defect in the gene for a-nicotinic acetylcholine receptor, CHRNA 3/5 (a chemical messenger that has also been linked to smoking and lung cancer) are associated with COPD. Alpha-1 antitrypsin deficiency (A1AD) is a rare genetic risk factor associated with emphysema.

DIAGNOSIS

The diagnosis of COPD is based on a combination of factors, including smoking history, symptoms, such as, wheezing, shortness of breath, physical examination, and spirometry results. A range of other symptoms includes poor exercise tolerance, productive or non-productive cough, respiratory failure, and cor pulmonale. Post-bronchodilator results demonstrate airflow limitation with diminished forced expiratory volume in 1 second (FEV1) to Forced Vital Capacity (FEV1/FVC) or FEV1 to Vital Capacity (FEV1/VC) in a ratio of 0.70 or less. During the process of diagnosing COPD, the effect of acute exacerbations, such as environmental irritants, respiratory infections and seasonal/weather change, should be considered. Other diseases may present with similar symptoms including asthma, acute bronchitis, bronchiectasis, and lung cancer.

COPD negatively impacts quality of life. Hypoxemia can impair mental function and short-term memory. Emphysema is associated with a poor nutritional status, weight loss, and poor muscle mass. These weight changes contribute to poor health outcomes.

Spirometry results help to categorize disease stage: [5]

- Stage I (mild): FEV_1 80% or greater of predicted
- Stage II (moderate): FEV_1 50-79% of predicted
- Stage III (severe): FEV_1 30-49% of predicted
- Stage IV (very severe): FEV_1 less than 30% of predicted or FEV_1 less than 50% and chronic respiratory failure

TREATMENT & MANAGEMENT

There is no cure for COPD. However, treatment can be helpful, including lifestyle changes, inhaler medication, oxygen therapy and pulmonary rehabilitation. The goals of COPD treatment are to provide symptomatic relief, retard progression or disease, prevent exacerbations, reduce hospitalizations and mortality, and improve exercise tolerance and quality of life. There is no evidence that spirometric screening of asymptomatic patients is beneficial.

Stage I (mild): FEV_1 80% or greater of predicted	Short-acting bronchodilator as needed
Stage II (moderate): FEV_1 50-79% of predicted	Short-acting bronchodilator as needed Long-acting bronchodilator(s) Respiratory anticholinergics Cardiopulmonary rehabilitation for symptomatic patients unresponsive to inhaler treatment
Stage III (severe): FEV_1 30-49% of predicted and chronic respiratory failure	Mono or dual therapy Short-acting bronchodilator as needed Long-acting bronchodilator(s)
	Respiratory anticholinergics Inhaled glucocorticoids if repeated exacerbations; Cardiopulmonary rehabilitation
Stage IV (very severe): FEV_1 less than 30% of predicted or FEV_1 less than 50% and chronic respiratory failure	Short-acting bronchodilator as needed Long-acting bronchodilator(s) Cardiopulmonary rehabilitation Respiratory anticholinergics Inhaled glucocorticoids if repeated exacerbation Long-term oxygen therapy Consider surgical options such as lung volume reduction surgery (LVRS) and lung transplantation

COPD TREATMENTS

The medications target potentially reversible causes of airflow limitation, such as reduction of bronchial smooth muscle contraction, mucosal congestion and edema, airway inflammation, and secretions. Potential adverse effects of treatment include oropharyngeal candidiasis and moderate to severe easy bruisability with inhaled corticosteroids, mouth dryness with tiotropium, and increased cardiovascular events with long-acting inhaled β-agonists. Inhaled corticosteroids are recommended in patients at increased likelihood of exacerbations; however, they can be safely withdrawn in patients with moderate COPD and no history of frequent exacerbation, while maintaining adequate bronchodilator therapy.[6]

Short-acting $beta_2$ -agonist (albuterol, metaproterenol, levalbuterol, pirbuterol)

- Long-acting $beta_2$ -agonist (salmeterol, formoterol, arformoterol, indacaterol, vilanterol)
- Respiratory anticholinergics (ipratropium, tiotropium (spiriva), aclidinium)
- Xanthine derivatives (theophylline)
- Phosphodiesterase-4 Inhibitors (roflumilast)
- Inhaled corticosteroids (fluticasone, budesonide)
- Oral corticosteroids (prednisone)
- $Beta_2$ -agonist and anticholinergic combinations (ipratropium and albuterol, umeclidinium bromide/vilanterol inhaled)
- $Beta_2$ -agonist and corticosteroid combinations (budesonide/formoterol, fluticasone and salmeterol, vilanterol/fluticasone inhaled)

Pulmonary rehabilitation programs are typically multidisciplinary approaches that include family involvement, smoking cessation, optimization of medical management, physiotherapy, physical therapy and psychosocial support.

Lung Volume Reduction Surgery (LVRS) removes bullous areas in the upper lobes, thus reducing “dead space” and improving air movement through functional tissue. This may result in better breathing.

RECOMMENDATIONS

- Diagnose airflow obstruction with formal Pulmonary Function Tests including ABG's for symptomatic patients
- Baseline CXR
- Smoking cessation
- Pulmonary consultation for persistent symptoms despite treatment
- Administer Influenza vaccine.
- Administer pneumococcal vaccines (PPSV23 and PV13) according to national guidelines.
- Order Low Dose-CT of patients with COPD (who have a 30 pack year history and are 55-74 years old, who are either current smokers or quit within the past 15 years)[5]

SOURCES

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