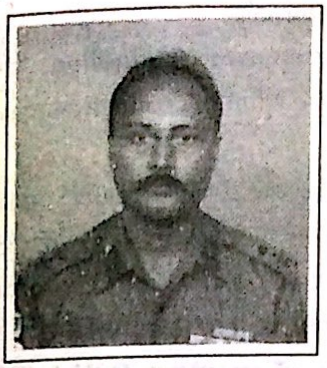
Update on Chronic Obstructive Pulmonary Disease



Lt. Col. (Dr.) B. Srivastava, MD
HOD Department of Chest Diseases.
Brig Gen (Dr.) Ramesh B. Basnyat
Registrar, SBH, Advisor for Chest Diseases

Chronic Obstructive Pulmonary Disease (COPD) is a major public health problem. It is the fourth leading cause of chronic morbidity and mortality in the United States and is projected to rank fifth in 2020 as a worldwide burden of disease according to a study published by the World Bank/World Health Organization. In Nepal, COPD ranks higher than expected due to various causes like exposure to smoke from biomass fuels in ill ventilated dwellings, early age of onset of smoking, outdoor air pollution and decreased awareness among people. Yet, COPD fails to receive adequate attention from the health care community and government officials. With these concerns in mind, a committed group of scientists encouraged the US National Heart, Lung, and Blood Institute (NHLBI) and the World Health Organization (WHO) to form the **Global Initiative for Chronic Obstructive Lung Disease (GOLD).**

Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. The Global Initiative for Chronic Obstructive Lung Disease was created to increase awareness of COPD among health professionals, public health authorities, and the general public, and to improve prevention and management through a concerted worldwide effort. The Initiative prepares scientific reports on COPD, encourages dissemination and adoption of the reports, and promotes international collaboration on COPD research.

This update is compiled from GOLD which is a Consensus Workshop Report by a distinguished group of health professionals from the fields of respiratory medicine, epidemiology, socioeconomics, public health, and health education, reviewed existing COPD guidelines, as well as new information on pathogenic mechanisms of COPD as they developed a consensus document in 2001.

Chronic Obstructive Pulmonary Disease (COPD) is a disease state characterized by 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. The most important risk factor for COPD is cigarette smoking. Pipe, cigar, and other types of tobacco smoking popular in many countries are also risk factors for COPD. At every possible opportunity individuals who smoke should be encouraged to quit. A diagnosis of COPD should be considered in any individual with symptoms and a history of exposure to risk factors. The diagnosis should be confirmed by spirometry.

This definition does not use the terms chronic bronchitis and emphysema and excludes asthma (reversible airflow limitation).

Symptoms of COPD include:

- Cough
- Sputum production
- Dyspnea on exertion.

Episodes of acute worsening of these symptoms often occur. **Chronic cough and sputum production often precede the development of airflow limitation by many years, although not all individuals with cough and sputum production go on to develop COPD.**

Chronic bronchitis, defined as the presence of cough and sputum production for at least 3months in each of 2 consecutive years, is not necessarily associated with airflow limitation.

Emphysema, defined as destruction of the alveoli, is a pathological term that is sometimes (incorrectly) used clinically.

Risk factors: what causes COPD ?

Tobacco Smoke: The most important risk factor for COPD is cigarette smoking. Pipe, cigar, and other types of tobacco smoking popular in many countries are also risk factors for COPD.

Other documented causes of COPD include: Occupational dusts and chemicals (vapors, irritants, and fumes), indoor air pollution from biomass fuel, outdoor air pollution, which adds to the lungs' total burden of inhaled particles, passive exposure to cigarette smoke also contributes to respiratory symptoms and COPD. Respiratory infections in early childhood are associated with reduced lung function and increased respiratory symptoms in adulthood.

DIAGNOSING COPD

A diagnosis of COPD should be considered in any individual who presents with characteristic symptoms and a history of exposure to risk factors for the disease, especially cigarette smoking.

Key Indicators for Considering a Co Diagnosis

- **Chronic cough:** Present intermittent every day, often throughout the day sel only nocturnal.
- **Chronic sputum production:** Any pa of chronic sputum production may ind COPD.
- **Acute bronchitis:** Repeated episodes
- **Dyspnea** that is progress persistent, worse on exercise during respiratory infections.
- **History of exposure to risk factors**

The diagnosis should be confirmed by spirome

Patients with COPD typically show a decrea both FEV1 and FEV1/FVC. The degre spirometric abnormality generally reflects the seve of COPD.

Classification of COPD by severity

Stage 0: At Risk - Chronic cough and spu production; lung function is still normal.

Stage I: Mild COPD - Mild airflow limita (FEV1/FVC < 70% but FEV1 is > 80 % predic and usually, but not always, chronic cough sputum production. At this stage, the individual not be aware that his or her lung functio abnormal.

Stage II: Moderate COPD - Worsening airf limitation, progression of sympto (30% > FEV1 < 80% predicted). Acute exacer tions of symptoms, which have an impact o patient's quality of life and prognosis, are especi seen in patients with FEV1< 50% predicted.

Stage III: Severe COPD - Severe airfl limitation, with respiratory failure or clinical sign right heart failure, (FEV1 < 30% predicted) or e if the FEV1 is > 30% predicted, whenever th

complications are present. At this stage, quality of life is very appreciably impaired and exacerbations may be life-threatening.

Differential Diagnosis:

A major differential diagnosis is asthma. In some patients with chronic asthma, a clear distinction from COPD is not possible using current imaging and physiological testing techniques. In these patients, current management is similar to that of asthma. Other potential diagnoses are usually easier to distinguish from COPD.

Differential Diagnosis of COPD

COPD: Onset in mid-life, symptoms slowly progressive, long smoking history, dyspnea during exercise, irreversible airflow limitation.

Asthma: Onset early in life (often childhood), symptoms vary from day to day more at night/early morning, reversible airflow limitation. Allergy, rhinitis, and/or eczema also present. Family history of asthma.

Congestive Heart Failure: Fine basilar crackles on auscultation. Chest X-ray shows dilated heart, pulmonary edema. Pulmonary function tests indicate volume restriction, not airflow limitation.

Bronchiectasis: Large volumes of purulent sputum, commonly associated with bacterial infection, coarse crackles/clubbing on examination. Chest X-ray/CT shows bronchial dilation, bronchial wall thickening.

Tuberculosis: Onset all ages, chest X-ray shows lung infiltrate or nodular lesions. Microbiological confirmation. High local prevalence of tuberculosis.

A person who has never smoked may develop COPD in the developing world, where other risk factors may be more important than smoking. Asthma may develop in adult and even elderly patients.

A COPD MANAGEMENT PROGRAM

The goals of COPD management include:

- Prevent disease progression
- Relieve symptoms
- Improve exercise tolerance
- Improve health status
- Prevent and treat complications
- Prevent and treat exacerbations
- Reduce mortality
- Prevent or minimize side effects from treatment.

Cessation of cigarette smoking should be included as a goal throughout the management program. These goals can be achieved through implementation of a COPD management program with four components:

Component 1: Assess And Monitor Disease

A detailed medical history of a new patient known or thought to have COPD should assess exposure to risk factors, past medical history of asthma, allergy, sinusitis or nasal polyps, respiratory infections, and other respiratory diseases, history of exacerbations, presence of comorbidities, such as heart disease and rheumatic disease. Impact of disease on patient's life, including limitation of activity; missed work and economic impact; effect on family routines; and feelings of depression or anxiety. Possibilities for reducing risk factors, especially smoking cessation. In addition to **spirometry**, the following **other tests** should be undertaken for the assessment of a patient with Moderate to Severe COPD (Stage II or III):

- **Bronchodilator reversibility testing:** To rule out a diagnosis of asthma and guide initial treatment decisions.
- Inhaled glucocorticosteroid trial (6 weeks to 3 months):

To identify patients with airflow limitation that is responsive to inhaled glucocorticosteroid treatment. If objective benefit is not demonstrated, ICS should be discontinued.

• **Chest X-ray:** Seldom diagnostic in COPD but valuable to exclude alternative diagnoses, e.g., pulmonary tuberculosis.

Arterial blood gas measurement: In patients with FEV1 < 40% predicted or with clinical signs suggestive of respiratory failure or right heart failure.

• **Alpha-1 antitrypsin deficiency screening:** Perform when COPD develops in patients under 45 years, or in patients with a strong family history of COPD.

COPD is usually a progressive disease. Lung function can be expected to worsen over time, even with the best available care. Symptoms and lung function should be monitored to follow the development of complications, to guide treatment, and to facilitate discussion of management options with patients.

Component 2: Reduce Risk Factors

Smoking cessation is the single most costeffective intervention to reduce the risk of developing COPD and slow its progression.This can beacheived by counseling and Pharmacotherapy (nicotine replacement and/or buproprion).

Smoking Prevention: Encourage comprehensive tobacco-control policies and programs with clear, consistent, and repeated nonsmoking messages. Work with government officials to pass legislation to establish smoke-free schools, public facilities, and work environments and encourage patients to keep smoke-free homes.

Occupational Exposures: Emphasize prim prevention, which is best achieved by eliminatio reduction of exposures to various substances in workplace.

Indoor and Outdoor Air Pollution: Implem measures to reduce or avoid indoor air pollution biomass fuel, burned for cooking and heating in po ventilated dwellings.

Component 3: Manage Stable COPD

General principles:

1. Determine disease severity.
2. Implement a stepwise treatment plan.
3. Choose treatments according national and cultural preferences.
4. Patient education.
5. Pharmacologic treatment:

Bronchodilators

Give "as-needed" to relieve intermitten worsening symptoms and on a regular basi prevent or reduce persistent symptoms. The ch between drugs depends upon patient's indivi response in terms of both symptom relief and effects. Regular treatment with short-ac bronchodilators is cheaper but less convenient treatment with long-acting bronchodilators.

Pharmacologic treatment can impr and prevent symptoms, reduce frequency and severity of exacerbati improve health status, and improve exer tolerance.Combining drugs with diffe mechanisms and durations of action increase the degree of bronchodilation equivalent or lesser side effe **Theophylline is effective in COPD, but d its potential toxicity inhaled bronchodilator preferred when available.**

Regular nebulized bronchodilator therapy for a s patient is not appropriate unless it has been sh

to be better than conventional doses by metered dose inhaler.

Glucocorticosteroids: Regular treatment with inhaled glucocorticosteroids is only appropriate for patients with:

- symptomatic improvement and a documented spirometric response to inhaled glucocorticosteroids or
- an FEV1 < 50% predicted and repeated exacerbations requiring treatment with antibiotics or oral glucocorticosteroids.

Prolonged treatment with inhaled glucocorticosteroids may relieve symptoms in **this carefully selected group of patients** but does not modify the long-term decline in FEV1. **Long-term treatment with oral glucocorticosteroids is not recommended.**

Vaccines: *Influenza* vaccines reduce serious illness and death in COPD patients by 50%. Give once in autumn or twice in autumn and winter each year. There is no evidence for recommending the general use of pneumococcal vaccine for COPD.

Antibiotics: Recommended only for treatment of exacerbations and other bacterial infections.

Mucolytic (Mucokinetic, Mucoregulator) Agents: Not recommended.

Antitussives: Regular use contraindicated in stable COPD.

Respiratory Stimulants: Not recommended for regular use.

Oxygen Therapy: The long-term administration of oxygen (>15 hours per day) to patients with chronic respiratory failure increases survival and has a beneficial impact on pulmonary arterial pressure, polycythemia, exercise capacity, lung mechanics, and mental state. The goal of oxygen therapy is to increase the baseline PaO2 at rest to at least 8.0 kPa (60 mm Hg) at sea level, and/or produce SaO2 at least 90%, which will preserve vital organ function by ensuring an adequate delivery of oxygen. Initiate oxygen therapy for patients with Severe COPD (Stage III) if:

- PaO2 is at or below 7.3 kPa (55 mm Hg) or SaO2 is at or below 88%, with or without hypercapnia; or
- PO2 is between 7.3 kPa (55 mm Hg) and 8.0 kPa (60 mm Hg) or SaO2 is 89%, if there is evidence of pulmonary hypertension, peripheral edema suggesting congestive heart failure, or polycythemia.

Surgical Treatments:

Bullectomy and lung transplantation may be considered in carefully selected patients with severe COPD (Stage III). There is currently no sufficient evidence that would support the widespread use of lung volume reduction surgery (LVRS).

There is no convincing evidence that mechanical ventilatory support has a role in the routine management of stable COPD.

Component 4: Manage Acute Exacerbations

COPD is often associated with acute exacerbations of symptoms. Many exacerbations are caused by infection of the tracheobronchial tree or an increase in air pollution, but the cause of about one-third of severe exacerbations cannot be identified. A person with COPD has an acute exacerbation when he has the following symptoms:

1. worsening dyspnoea.
2. cough with increased sputum production.
3. increased sputum purulence.

Acute exacerbations can be graded as follows:

- **Type I(severe)** - All 3 symptoms
- **Type II(moderate)** - 2 symptoms
- **Type III(mild)** - At least 1 symptom + 1 of the clinical criteria
  * URTI within 5 days
  * Fever without apparent cause
  * Increased wheezing
  * Increased cough
  * Increased RR or HR by >20% of baseline

How to Assess the Severity of an Acute Exacerbation?

- PEF < 100 L/min or FEV1 < 1 L indicates a severe exacerbation.
- PaO2 < 8.0 kPa (60 mm Hg) and/or SaO2 < 90% when breathing room air indicate respiratory failure.
- PaO2 < (50 mm Hg), PaCO2 > (70 mm Hg), and pH < 7.30 suggest a life-threatening episode that needs close monitoring or ICU management.

Chest X-ray: Chest radiographs (posterior/anterior plus lateral) identify complications such as pneumonia, pneumothorax and other conditions that mimic an acute exacerbation.

ECG: Aids in the diagnosis of right ventricular hypertrophy, arrhythmias, and ischemic episodes.

Other laboratory tests:

- Sputum Gram stain and culture.
- Biochemical tests to detect electrolyte disturbances, diabetes, and poor nutrition.

Home Management

Bronchodilators: Increase dose and/or frequency of existing bronchodilator therapy. If not already used, add anticholinergics until symptoms improve.

Glucocorticosteroids: If baseline FEV1 < 50% predicted, add 40 mg oral prednisolone per day for 10 days to the bronchodilator regimen.

Antibiotics: When symptoms of breathlessness, cough are increased and sputum is purulent, increased in volume, provide antibiotic coverage the major bacterial pathogens involv inexacerbations, taking into account local patte of antibiotic sensitivity.

Hospital Management

Patients with the following characteristics shoul considered for hospitalization:

- Marked increase in resting dyspnea, onse new physical signs (e.g., cyano peripheral edema).
- Failure of exacerbation to respond to in medical management.
- Significant comorbidities.
- Newly occurring arrhythmias, diagnos uncertainty.
- Older age, insufficient home support.

Indications for ICU Admission

Severe dyspnea, that responds inadequately to in emergency therapy. Confusion, lethargy and co Persistent or worsening hypoxemia (PaO2 < 50 Hg), worsening hypercapnia (PaCO2 > 70 mm H and/or severe/worsening respiratory acido (pH < 7.30) despite supplemental oxygen and NIP

Indications for Mechanical Ventilatio

1. Severe dyspnea with use of access muscles and paradoxical abdominal moti
2. Respiratory frequency > 35 breaths minute.
3. Life-threatening hypoxemia (PaO2 < 40 Hg or PaO2/FiO2 < 200.
4. Severe acidosis (pH < 7.25) hypercapnia (PaCO2 > 60 mm Hg).
5. Respiratory arrest.
6. Somnolence, impaired mental status.
7. Cardiovascular complications (hypotens shock, heart failure).
8. Other complications (metabo abnormalities, sepsis, pneumo pulmonary embolism, barotrauma, mass pleural effusion).
9. NIPPV failure.

Discharge Criteria for Patients with Acute Exacerbations of COPD

1. Inhaled ß2-agonist therapy is required no more than every 4 hrs.
2. Patient, if previously ambulatory, is able to walk across room.
3. Patient is able to eat and sleep without awakening by dyspnea.
4. Patient has been clinically stable for 12-24 hrs.
5. Arterial blood gases have been stable for 12-24 hrs.
6. Patient fully understands correct use of medications.
7. Follow-up and home care arrangements have been completed.
8. Patient, family, and physician are confident patient can manage.

Future Rasearch

A better understanding of the molecular and cellular pathogenic mechanisms of COPD should lead to many new directions for both basic and clinical investigations. Improved methods of early detection, new approaches for interventions through targeted pharmacotherapy, possible means to identify the "susceptible" smoker, and more effective means of managing exacerbations are needed.

Until there is a better understanding of the causal mechanisms of COPD, an absolutely rigid definition of COPD, and its relationship to other obstructive airways diseases, will remain controversial.

Surrogate markers of inflammation, possibly derived from the analysis of sputum (cells, mediators, enzymes) or exhaled condensates (lipid mediators, reactive oxygen species, cytokines), that may predict the clinical usefulness of new management and prevention strategies for COPD need to be developed.

The cellular and molecular mechanisms responsible for the persistence of the inflammatory response in stable COPD, should be investigated. Why inflammation in COPD is poorly responsive to glucocorticosteroids and what treatments other than glucocorticosteroids are effective in suppressing inflammation in COPD are research topics that could lead to new treatment modalities.

Standardized methods for tracking trends in COPD prevalence, morbidity, and mortality over time need to be developed so that countries can plan for future increases in the need for health care services in view of predicted increases in COPD.

Longitudinal studies demonstrating the course of COPD are needed in a variety of populations exposed to various risk factors. Factors that determine why some, but not all, smokers develop COPD need to be identified.

While spirometry is recommended to assess and monitor COPD reproducible and inexpensive exercise testing methodologies (e.g., stairclimbing tests) suitable for use in developing countries need to be evaluated.

Since COPD is not fully reversible and slowly progressive, it is important to identify early cases as more effective therapies emerge. Consensus on standard methods for detection and definition of early disease need to be developed. Data to show whether or not screening is effective in directing management decisions in COPD outcomes are required.

References

1. Global initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, And prevention of chronic obstructive pulmonary disease NHLBI/WHO workshop report (Based on an April 1998 meeting) National Institutes Of Health. National Heart, Lung, and Blood Institute publication number 2701 March 2001.
2. The evidence base for management of acute exacerbations of COPD. Snow V., Lascher S., Mottur-Pilson C. Annals of Internal Medicine 2001; 134: 595-599.
3. Management of acute exacerbations of COPD. A summary and appraisal of published evidence. McGory D.C., Brown C., Gelfand B.A., Bach P.B. Chest(India Edition) 2001; 2(No. 4): 205-224.

❑❑❑