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REVIEW

# Management of the patient with chronic obstructive airway disease (COPD) in a primary health care context

**I Govender,[1] HI Okonta,[2] S Rangiah,[3] D Nzaumvila[2]**

*[1] Department of Family Medicine, University of Pretoria, South Africa*
*[2] Department of Family Medicine, Sefako Makgatho Health Sciences University, South Africa*
*[3] Department of Family Medicine, University of KwaZulu-Natal, South Africa*
*Corresponding author, email: indiran.govender@gmail.com*

This paper describes the incidence of chronic obstructive airway disease (COPD), the risk factors, staging, investigations and management of COPD. The differential diagnosis for COPD is also presented as COPD can be confused with other clinical conditions. This paper is presented in practical terms for the clinician working in a primary health care context.

## Introduction

Chronic obstructive airway disease (COPD) is a progressive lung disease characterised by airflow obstruction that is not fully reversible and is associated with abnormal inflammatory responses of the lung to noxious particles or gases. The global prevalence of COPD is between 7–19%, with variations in different regions of the world, and a predominance in men and people aged over 40 years.[1] In Cape Town, South Africa, the prevalence of COPD is 22.2% for men and 16.7% for women.[1] Mintz et al. have determined that 21% of patients aged 30 years or more with more than 10 years of smoking history seen in primary care settings are likely to have COPD.[2] The Global Burden of Disease Study 2010 reported a prevalence estimate of 8.9%[3] and it is expected that it will be the 6th cause of death in the world with an increase in economic and social burden.[4]

## Risk factors

Tobacco use is the most important cause and contributing factor for COPD progression. Occupational exposures from gold mining, coal mining and cotton textile dust, indoor air pollution from burning wood and other biomass fuels, and poorly burning stoves also play a causative role. In addition, infections such as childhood respiratory infections and pulmonary TB, airway hypersensitivity, α1-antitrypsin deficiency, and demographic factors such as age, family history of atopy and low socioeconomic status, all contribute to the development of COPD.

## Clinical presentation

The two subtypes of COPD – chronic bronchitis and emphysema – often coexist with their pathologic and clinical features (Table 1).[5] The onset of COPD is insidious, with chronic cough, expectoration, and exertional dyspnoea. A high index of suspicion should be considered in every patient with risk factors of the disease.[4]

**Table 1.** Features distinguishing chronic bronchitis from emphysema

| Features | Chronic bronchitis (Blue bloaters) | Emphysema (Pink puffers) |
|---|---|---|
| Definition | Definition is clinical: Productive cough on most days for at least 3 consecutive months in 2 successive years | Definition is pathological: Dilatation and destruction of air spaces distal to the terminal bronchiole without obvious fibrosis |
| Pathology | Obstruction due to narrowing of the airway lumen by mucosal thickening and excess mucus | Decreased elastic recoil of lung parenchyma due to air trapping, airway collapse and decreased expiratory driving pressure<br>In smokers the lesions are centriacinar primarily affecting the upper lung zones<br>In patients with α1-antitrypsin deficiency the lesions are panacinar primarily affecting the lower lobes |
| Clinical presentation | Symptoms: Chronic productive cough, purulent sputum<br>Signs: Cyanosis, peripheral oedema, crackles, wheezes, prolonged expiration, obesity | Symptoms: Exertional dyspnoea, minimal cough, tachypnoea, decreased exercise tolerance<br>Signs: Pink skin, pursed-lip breathing, use of accessory muscles, cachexia, barrel chest, hyperresonant percussion, decreased breath sounds, decreased diaphragmatic excursion |
| Investigations | PFT: ↓$FEV_1$, ↓$FEV_1$/FVC, normal TLC, ↓or normal $DL_{co}$<br>CXR: Normal AP diameter, ↑bronchovascular markings, enlarged heart with cor pulmonale | PFT: ↓$FEV_1$, ↓$FEV_1$/FVC, ↑TLC, ↑RV, ↑$DL_{co}$<br>CXR: ↑AP diameter, flat hemidiaphragm (on lateral x-ray, ↓cardiac silhouette, ↑retrosternal space, bullae, ↓peripheral markings) |

PFT = Pulmonary function tests; $FEV_1$=Forced expiratory volume in 1 second; FVC=Forced vital capacity; TLC=Total lung capacity; $DL_{co}$=Carbon monoxide diffusing capacity of lung

During acute exacerbations, patients may present with fever, chest pain and severe shortness of breath. Physical examination may reveal respiratory distress with use of accessory muscles and intercostal recession, decreased chest expansion, barrel chest with hyper-resonant percussion, distant breath sounds and poor air entry on auscultation. There may be crackles and wheezes with prolonged expiration. The presence of cyanosis and asterixis may indicate respiratory failure secondary to hypoxemia and hypercapnia. Distended neck veins, hepatomegaly with hepatojugular reflux and bilateral pedal oedema are suggestive of cor pulmonale.

The severity of dyspnoea in COPD can be assessed using the Modified British Medical Research Council (MMRC) scale[4] (Table 2).

**Table 2.** Modified British Medical Research Council Dyspnoea scale

| Scale | Severity of dyspnoea |
|---|---|
| 0 | No breathlessness except with strenuous exercise |
| 1 | Shortness of breath when hurrying on the level or walking up a slight hill |
| 2 | Walks slower than people of the same age on the level because of breathlessness or has to stop for breath when walking at own pace on the level |
| 3 | Stops for breath after walking about 100 metres or after a few minutes on the level |
| 4 | Too breathless to leave the house or breathless when dressing or undressing |

## Investigations

- Spirometry is used to determine the extent of airflow obstruction, assess the severity of COPD and monitor the disease progression. Spirometry is not recommended in acute exacerbation as this may be difficult to perform and may not be accurate. A post-bronchodilator $FEV_1$/FVC of < 0.70 is diagnostic of COPD.
- The severity of airflow obstruction is assessed using the Global Initiative for Chronic Obstructive Lung Disease (GOLD)[4] criteria. (Table 3)

**Table 3.** COPD severity grading – GOLD criteria

| Grade | $FEV_1$/FVC | $FEV_1$ |
|---|---|---|
| Mild COPD (GOLD 1) | < 0.7 | ≥ 80% of predicted |
| Moderate COPD (GOLD 2) | < 0.7 | < 80% but ≥ 50% of predicted |
| Severe COPD (GOLD 3) | < 0.7 | < 50% but ≥ 30% of predicted |
| Very severe COPD (GOLD 4) | < 0.7 | < 30% of predicted or $FEV_1$ < 50% with respiratory failure or right-sided heart failure |

- Chest x-ray is done to exclude other conditions or identify co-morbidities.

## Radiographic signs of hyperinflation are:

- Increased anteroposterior diameter, increased retrosternal airspace, reduced cardiac silhouette with a vertical heart, flattening of the diaphragms, (better demonstrated on lateral chest x-ray), enlarged central pulmonary arteries and reduced peripheral vascular markings.
- Chest CT scan can demonstrate emphysema, cystic and bullous lesions.
- Echocardiogram is recommended if the physical examination findings are suggestive of cor pulmonale.
- Full blood count may show polycythaemia due to hypoxia or anaemia
- Eosinophil count > 100 cells/uL in recurrent exacerbations determines need for inhaled corticosteroid[4]
- Blood gases may reveal hypoxia if peripheral oxygen saturation is < 92% or respiratory failure if PCO2 > 45
- Serum $\alpha_1$-antitrypsin level is indicated in patients aged ≤ 45 years with minimal or no smoking history, positive family history of COPD and lower lobe emphysema.

## Differential diagnosis

The differential diagnosis of a patient with dyspnoea and persistent productive cough includes the following conditions:

- **Asthma**

  Begins in childhood and is usually episodic. There may be personal or family history of allergy, eczema or rhinitis. The episodes are characterised by wheezing and the airway obstruction is reversible with bronchodilators.
- **Bronchiectasis**

  This may be difficult to differentiate from chronic bronchitis. The sputum is copious with occasional haemoptysis. There may be past history of pneumonia, pertussis, tuberculosis and nontuberculous mycobacterium infection. Chest x-ray finding of tram tracking (parallel narrow lines) radiating from the hilum and cystic spaces are suggestive of bronchiectasis. A high-resolution chest CT will show bronchial dilatation and bronchial wall thickening to confirm the diagnosis.
- **Pneumonia**

  Usually presents with fever, chills and pleuritic chest pain. Chest percussion is dull and auscultation may reveal crackles or bronchial breath sounds. The area of infiltration is confirmed by chest x-ray.
- **Tuberculosis**

  Usually manifests with chronic productive cough, fever, night sweats and weight loss. Chest x-ray may reveal infiltrates, cavitation or fibrosis. A positive sputum Acid Fast Bacilli (AFB), GeneXpert or culture confirms the diagnosis.
- **Congestive heart failure**

  Usually presents with orthopnoea and basilar crackles may be heard on auscultation. Brain natriuretic peptide level is elevated, chest x-ray shows cardiomegaly with increased pulmonary vascular congestion, and echocardiogram confirms the diagnosis.
- **Lung cancer**

  Patients with lung cancer may present with cough, haemoptysis, dyspnoea, chest pain, weight loss and night sweats. This needs to be considered as smoking is a risk

factor for lung cancer and COPD. The physical examination and chest x-ray or chest CT findings will suggest further diagnostic investigations (bronchoscopy, lung/pleural biopsy, thoracenthesis) towards tissue pathology diagnosis of lung cancer.

## Management

The management of COPD is based on the GOLD grade and exacerbation history (Table 4). Tobacco use must be explored and discussed with patients at all visits and if necessary motivational interviewing to stop tobacco use must be initiated as early as possible.[4]

There are management interventions that prolong survival[5] (Table 5). All other management interventions provide symptom relief with no mortality benefit.

The progress of COPD is fraught with gradual decrease in $FEV_1$ and episodes of acute exacerbations. Acute exacerbation of COPD is defined as sustained worsening of dyspnoea, cough, or sputum production of acute onset leading to increased use of medications. The most common trigger is viral or bacterial upper respiratory infection. Other causes include irritants from air pollution, pulmonary embolism, myocardial infarction, anaemia, and congestive heart failure.

The investigations in the setting of acute exacerbation of COPD include FBC, chest x-ray, ECG and pulse oximetry and/or arterial blood gas. Spirometry is not recommended in acute exacerbation as this may be difficult to perform and may not be accurate.

The management of acute exacerbation of COPD includes the following:

- The airway should be secured and ventilation assisted in patients with altered level of consciousness.

**Table 4.** Management of COPD based on GOLD grade and exacerbation history

| Patient group | Description | Non-pharmacological treatment | Pharmacological treatment |
|---|---|---|---|
| A | Few symptoms and low risk of exacerbation: MMRC grade 0–1 or CAT score < 10<br><br>0–1 exacerbations per year not requiring hospitalisation | Smoking cessation<br><br>Physical activity<br><br>Vaccinations | SAMA or SABA prn<br><br>SAMA + SABA prn<br>(Combination therapy is superior to monotherapy)[5] |
| B | More symptoms and low risk of exacerbation: MMRC grade ≥ 2 or CAT ≥ 10<br><br>0–1 exacerbation per year not requiring hospitalisation | Smoking cessation<br><br>Physical activity<br><br>Vaccinations<br><br>Pulmonary rehabilitation | LAMA or LABA<br><br>LAMA + LABA<br>(combination therapy is superior to monotherapy)[5-7] |
| C | Few symptoms but high risk of exacerbation: MMRC grade 0–1 or CAT < 10<br><br>≥ 2 exacerbations per year, or one or more requiring hospitalisation | Smoking cessation<br><br>Physical activity<br><br>Vaccinations<br><br>Pulmonary rehabilitation | LAMA<br><br>LAMA + LABA or<br><br>LABA + ICS<br>(ICS increases risk of pneumonia and consequently less preferred) |
| D | More symptoms and high risk of exacerbation: MMRC grade ≥ 2 or CAT ≥ 10<br><br>≥ 2 exacerbations per year, or one or more requiring hospitalisation | Smoking cessation<br><br>Physical activity<br><br>Vaccinations<br><br>Pulmonary rehabilitation | LAMA + LABA<br><br>LAMA + LABA + ICS<br><br>LAMA + LABA +ICS + Roflumilast or Macrolide or stop ICS |

MMRC = Modified British Medical Research Council dyspnoea scale; SAMA = Short acting muscarinic antagonist; SABA = Short acting beta 2 agonist; LAMA = Long acting antimuscarinic antagonist; LABA = Long acting beta 2 agonist; ICS= Inhaled corticosteroid

**Table 5.** Management interventions that prolong survival

| Interventions | Details |
|---|---|
| Smoking cessation | The most effective intervention for preventing progression of COPD and includes:<br><br>Behavioural therapy, nicotine replacement, varenicline (Champix), bupropion and reinforcement of cessation at every contact |
| Vaccination | Influenza vaccination annually<br><br>Pneumococcal vaccination at least once per lifetime but can be repeated in 5–10 years |
| Home oxygen | Prevents cor pulmonale and decreases mortality if used > 15 hours/day<br><br>Indications include:<br>• $PaO_2 \leq 55$ mmHg<br>• $PaO_2 \leq 60$ mmHg with cor pulmonale or haematocrit > 55% |

- Oxygen is administered to maintain the $SaO_2$ between 88–92% for $CO_2$ retainers.
- Bronchodilators: Escalated use of SAMA + LAMA delivered by metered-dose inhaler (MDI) with spacer or nebuliser.
- Systemic corticosteroids: Oral prednisone 40 mg daily, for 5–7 days without tapering. Alternatively, IV methylprednisolone 125 mg 12 hourly could be used but this has increased risk of side effects.
- Antibiotics: These are indicated for exacerbations with increased sputum production plus either increased dyspnoea or increased sputum purulence. The choice of antibiotics is based on patient risk factors and local antibiotic resistance patterns as follows[5,8]:
  1. For simple exacerbation with no risk factors ($FEV_1$ > 50% predicted, ≤ 4 exacerbations/year, no cardiac disease): 5-day course of Amoxicillin, Macrolide, Trimethoprim/sulphamethoxazole, 2nd and 3rd generation cephalosporins.
  2. For complicated exacerbation ($FEV_1$ < 50% predicted, ≥ 4 exacerbations/year, cardiac disease): 7–10 day course of Amoxicillin-clavulanate, levofloxacin or moxifloxacin.
  3. For patients at risk of pseudomonas infection ($FEV_1$ < 35% predicted, chronic steroid use, persistent purulent sputum): Ciprofloxacin.

## Prognosis

The prognosis of COPD is variable, depending on genetic predisposition, environmental exposures, comorbidities and acute exacerbations. The single best prognostic predictor is the level of dyspnoea. The BODE index is a 10-point measure of four factors (body mass index, obstruction, dyspnoea and exercise capacity) used to predict mortality in COPD (Table 6). The probability that the patient will die from COPD increases as the BODE index score increases.

**Table 6.** BODE index score to predict mortality in COPD[4]

| Prognostic factor | BODE index score | | |
|---|---|---|---|
| | + 1 point | + 2 points | + 3 points |
| Body mass index (BMI) | ≤ 21 | | |
| Obstruction ($FEV_1$) | 50–64% | 36–49% | ≤ 35% |
| Dyspnoea (MMRC scale) | 2 | 3 | 4 |
| Exercise capacity (6-minute walk distance) | 250–349 metres | 150–249 metres | ≤ 149 metres |

Another prognostic tool that is sometimes used is the codex index which uses 4 factors (comorbidities, obstruction, dyspnoea and previous severe exacerbations) to predict the course of COPD.

## Follow-up

Patients with COPD should be followed up regularly to monitor disease progression or development of complications. Stable COPD patients can be followed up at three to six monthly intervals while patients with severe frequent exacerbations or recently discharged from hospitalisation should be followed up at two to four weekly intervals.

At each follow up visit, determine the level of dyspnoea at rest and with exercise, the number of exacerbations and hospitalisations. The COPD assessment test (CAT) questionnaire can be used for monitoring. Smoking cessation or avoidance of air pollution should be reinforced. Assess the effectiveness of medications and side effects. If medication appears ineffective, observe inhaler technique to ensure optimal delivery of current medications before adjustments. Oxygen saturation should be determined to identify COPD patients in need of supplemental oxygen. Annual spirometry is recommended to monitor decline in pulmonary function.

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