

Chronic obstructive pulmonary disease: management of chronic disease

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Abstract

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide, affecting an estimated 3 million people in the UK. The most common cause is tobacco smoke. Patients with COPD experience a high symptom burden, worsened during disease instability (termed exacerbations or 'lung attacks'), and a multidisciplinary approach should be adopted to manage this chronic lung disease. Diagnosis requires clinical and functional assessment to tailor treatments towards symptoms; the most common and debilitating of these are breathlessness, cough and sputum production. Breathlessness develops as a result of irreversible airway narrowing (obstruction), and spirometry is used alongside imaging to guide both diagnosis and treatment. To date, smoking cessation is the single most important intervention in delaying disease progression and should be a focus at every patient interaction. COPD is treated by a combination of pharmacological and non-pharmacological treatments, including pulmonary rehabilitation and self-management plans, allowing control over some of the symptom burden. Holistic management in COPD requires effective communication between all those involved in patient care, crossing secondary and primary care boundaries.

Keywords Chronic obstructive pulmonary disease; multidisciplinary team; pulmonary rehabilitation; smoking cessation

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Key points

- COPD is a major cause of morbidity and mortality in the UK and is increasing in prevalence
- The aims of therapy are to reduce symptoms, reduce risk of acute exacerbation and improve prognosis
- Therapy with inhaled medication is effective in improving symptoms and reducing exacerbation rates
- Non-drug therapies (smoking cessation, pulmonary rehabilitation, nutritional support and vaccination) are important in addressing the needs of patients
- End-of-life care and addressing advance care planning is essential to providing comprehensive care

Introduction

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide and the fifth highest in the UK. Approximately 1 million people in the UK have a diagnosis of COPD, with an estimated further 3 million of the population undiagnosed.

The disease occurs most commonly as a result of cigarette smoking, including second-hand smoke, but specific occupational exposure including biofuels can be a contributory cause. COPD is progressive, with irreversible lung damage leading to loss of lung function, physical decline and dependence on health and social care services. COPD is often punctuated by periods of worsening symptoms, termed exacerbations or 'lung attacks'. Exacerbations account for 1 in 8 of all emergency hospital admissions and 1 million 'bed-days' each year, which costs the British National Health Service about £500 million per year. The main aim of both secondary and primary care, through both pharmacological and non-pharmacological interventions, is to reduce and control exacerbations, and thus improve symptom burden and quality of life for patients with COPD. This heterogeneous disease requires individualized treatment incorporating a wide multidisciplinary team of clinicians, specialist nurses, physiotherapists, pharmacists, nutritionists, psychologists and palliative care.

Diagnosis

COPD is a broad disease label that encompasses several symptoms, patients often presenting with a combination of dyspnoea, predominately on exertion, cough and sputum production. Tobacco smoke is the most common cause, but patients should be asked about exposure to other inhaled substances, such as cannabis and biomass fumes. Other symptoms, such as weight loss, waking at night, ankle swelling and fatigue, should also be considered. Classification of symptoms using the Medical Research Council (MRC) Dyspnoea Scale (Table 1),¹ or COPD Assessment Test™ (CAT),² can be helpful in monitoring symptom progression.

Examination of patients with COPD may reveal few clinical signs, but the following should be recorded: presence or absence of cachexia, cyanosis, chest hyperinflation (Figure 1), pursed lip breathing, use of accessory muscles with respiration, and signs consistent with right heart failure and cor pulmonale, such as peripheral oedema and an elevated jugular venous pressure.

MRC Dyspnoea Scale¹

Grade	Degree of breathlessness related to activity
1	Not troubled by breathlessness except on strenuous exercise
2	Short of breath when hurrying or walking up a slight hill
3	Walks slower than contemporaries on level ground because of breathlessness, or has to stop for breath when walking at own pace
4	Stops for breath after walking about 100 metres or after a few minutes on level ground
5	Too breathless to leave the house, or breathless when dressing or undressing

Table 1

Investigations

COPD is characterized by irreversible airflow obstruction and classified using spirometry, measuring the post-bronchodilator forced expiratory volume in 1 second (FEV₁) and forced vital capacity (FVC). A post-bronchodilator FEV₁/FVC ratio <0.7 is consistent with a diagnosis of airflow obstruction. Disease severity is then classified according the FEV₁ deficit (Table 2).

To further manage and investigate patients with stable COPD, other routinely available investigations are necessary. These include: pulse oximetry, an oxygen saturation <92% saturation warranting referral for arterial blood sampling and a specialist oxygen assessment; a chest radiograph, to investigate the presence of bullae; and a full blood count to determine the presence of polycythaemia, which may merit referral on to secondary care for more detailed assessment. An α₁-antitrypsin concentration is warranted in all patients who present at a young age, have a positive family for this history or have a minimal smoking history (<20 pack years). In difficult-to-manage patients, where the diagnosis is uncertain or where there are significant exacerbations and symptoms, further specialist tests include: imaging (computed tomography; Figure 2); sputum culture for detailed microbiological sampling; full pulmonary function tests, which are typically associated with an elevated residual volume and reduced carbon monoxide diffusion capacity; and an echocardiogram to specifically assess elevated pulmonary artery pressure and right ventricular dysfunction.

Management of COPD

In stable disease, treatment focuses on reducing frequency of exacerbations and slowing disease progression. This is most effectively achieved through a combination of pharmacological and non-pharmacological therapies. These goals have been prioritized by both the British National Institute of Health and Care Excellence (NICE) COPD guidelines as well as the international Global Initiative for Chronic Obstructive Lung Disease (GOLD) recommendations. The latest revision of GOLD classifies patients according to a multidimensional assessment, taking into consideration lung function, risk of exacerbation and symptom burden (Figure 3). Treatment is then given according to the

individual’s disease phenotype. This approach is intuitive for physicians and patients alike, but it has yet to be underpinned by a robust evidence base demonstrating efficacy.

Pharmacological therapies

Inhaled therapy: the mainstay of inhaled treatment for COPD is maximal bronchodilation using both short- and long-acting β₂-adrenoreceptor agonists and anti-muscarinic agents, in addition to anti-inflammatories in the form of inhaled corticosteroids. Decisions on treatment relate to symptom burden, exacerbations and lung function (Figure 4). Treatment is often given in a stepwise approach, with most patients ultimately requiring triple inhaled therapy.

Emerging evidence has demonstrated distinct inflammatory phenotypes of COPD³ that could ultimately define tailored treatment for COPD. However, large clinical trials demonstrating this are still required.

Additional therapy: oral theophylline can be used in its slow-release form as add-on therapy in patients with COPD who continue to have symptoms despite inhaled therapy. Serum theophylline concentrations should be checked 3–5 days after starting treatment and then 1 week later. If stable they should be checked six-monthly. These medications are affected by cytochrome P450 enzyme inducers.

Oral mucolytic therapy can be trialled in patients with chronic productive cough and a high sputum load. They can help sputum expectoration and are often well tolerated.

Recent evidence has demonstrated that in patients with severe COPD and frequent exacerbations low-dose macrolide therapy is effective in reducing exacerbation frequency. The mechanisms behind this oral agent are still not understood.⁴ At this stage, low-dose macrolide therapy such agents cannot be recommended as add-on therapy outside secondary care.



Figure 1 Typical chest X-ray from a patient with COPD, showing gross hyperinflation, flattened hemi-diaphragms, horizontal rib configuration and a ‘stretched’ cardiac silhouette.

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