



Primary Care Strategies in Obstructive Airway Disease

DIAGNOSING AND MANAGING COPD

For Nurse Practitioners and Physician Assistants



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TARGET AUDIENCE

Nurse practitioners (NPs), physician assistants (PAs), and physicians in primary care practice.

ACTIVITY GOAL

To update NPs and PAs on practical strategies for recognizing chronic obstructive pulmonary disease (COPD) early and managing it according to evidence-based guidelines.

LEARNING OBJECTIVES

After completing this activity, participants should be better able to:

- Differentiate COPD from asthma based on information revealed by history, physical examination, and lung function testing
- Formulate treatment plans based on current practice guidelines and evidence-based strategies
- Implement monotherapies and combination therapies based on efficacy and safety data

ACCREDITATION INFORMATION



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Participants wishing to earn CME/CE credit must:

1. Read the newsletter.
2. Relate the content material to the learning objectives.
3. Complete the self-assessment questions and evaluation form online at www.practicingclinicians.com/testing/login.php. Successful completion of the self-assessment is required to earn CME/CE credit.

The estimated time to complete this activity is 1 hour.

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Please contact Practicing Clinicians Exchange at pce@cealliance.org for questions regarding this activity.

Chronic obstructive pulmonary disease (COPD), encompassing emphysema and chronic bronchitis, is characterized by airflow obstruction that may interfere with breathing at rest or on exertion.¹

COPD is one of the most common respiratory diseases in the United States. In 2007, an estimated 10.2 million adults self-reported a diagnosis of COPD (7.6 million with chronic bronchitis and 3.7 million with emphysema),² but according to national surveillance data, another 12 million adults were estimated to have a clinical profile of COPD, suggesting that COPD is grossly underdiagnosed.³ Women are twice as likely as men to be diagnosed with chronic bronchitis. In 2007, 5.0 million women were diagnosed with chronic bronchitis compared with 2.6 million men.²

The clinical and economic burden of COPD is staggering. Each year COPD is responsible for 15 million visits to clinician offices or hospital outpatient departments.⁴ In 2006, there were 672,000 COPD-related hospitalizations (22.5 per 10,000 population).¹ COPD was responsible for \$42.6 billion in total costs in 2007—\$26.7 billion in direct healthcare expenditures, \$8.0 billion in indirect morbidity costs, and \$7.9 billion in indirect mortality costs.⁴

COPD is a major cause of morbidity and mortality in the United States.^{1,3} Death rates from COPD have more than doubled since 1980 from 53,000 to 121,000 in 2006, representing 42 deaths per 100,000 population.¹ Although historically death rates had been higher in men than in women, since 2000 death rates in women have surpassed those in men.³ In 2005 (the most current year for which statistics are available), 51.9% of deaths due to COPD were in women.¹

COPD currently ranks as the fourth leading cause of death in the United States. By 2020, it is expected to be the third leading cause of death in the United States as well as globally.⁵

The clinical and economic burden of COPD underscores the importance of accurate diagnosis and appropriate clinical interventions. Guidelines for appropriate diagnosis and treatment are available.^{6,7} However, COPD continues to be underdiagnosed and undertreated or inappropriately treated, partly because it often is misdiagnosed as asthma.^{8,9} Women are more likely to be misdiagnosed as having asthma than men when presenting with similar COPD symptomatology.¹⁰ Although asthma and COPD share similarities in symptomatology and clinical presentation patterns, they differ in the stepwise approach to pharmacologic therapy. Misdiagnosis can lead to suboptimal care and increased morbidity.^{6,11}

This newsletter highlights the clinical and pathophysiologic differences between COPD and asthma and reviews current treatment paradigms for COPD.

CLINICAL CHARACTERISTICS: Differentiating COPD From Asthma

COPD and asthma are both inflammatory disorders characterized by a reduced rate of pulmonary airflow (airway obstruction), resulting from increased airway inflammation.¹² Symptoms and airflow obstruction usually are intermittent in asthma, whereas they are present con-

tinuously in COPD. In COPD, the airway obstruction is primarily structural/mechanical in nature. It is mostly due to increased secretions and sloughed cells that leads to a narrowing of the airway, smooth muscle contraction (largely due to increased cholinergic tone but also some muscle hypertrophy, although less than that in asthma), and premature collapse of airways during exhalation leading to air trapping with or without hyperinflation.⁶ In asthma, the airway obstruction is due to airway smooth muscle contraction, airway edema, airway thickening (due to structural changes in severe disease that is not fully reversible by current therapy), and mucus hypersecretion.¹³

Although COPD and asthma have similar symptoms—cough, dyspnea, and wheezing, there are distinct differences in patient profile, risk factors, clinical course, and pathophysiologic mechanisms (see *Inflammatory and Pathophysiologic Differences Between COPD and Asthma*, page 3).¹⁴ To correctly diagnose COPD, it is essential to consider the totality of these factors with spirometry findings.

Patient Profile

Age at onset of symptoms is an important distinguishing feature between COPD and asthma (Table 1).^{14,18} Individuals with COPD almost always present with symptoms after age 40 and predominantly have a >20 pack-year history of tobacco smoking. After age 40, the prevalence of COPD increases substantially with aging while the prevalence of patient-reported asthma declines with age.¹⁹ COPD in younger adults <45 years is rare, except in those with a history of alpha₁-antitrypsin deficiency or in those who are susceptible to tobacco smoke and begin significant smoking early or have diminished lung growth due to premature birth. In children, COPD essentially is unknown.¹ This clinical picture of a typical

Inflammatory and Pathophysiologic Differences Between COPD and Asthma

COPD and asthma differ in a number of pathophysiologic aspects including the anatomic sites of pathology, pathologic features, and underlying immunologic mechanisms.⁶

COPD affects the peripheral airways (premature collapse during exhalation leading to air trapping), proximal airways (bronchitis), lung parenchyma (emphysema), and pulmonary vessels (pulmonary hypertension),⁶ while in most patients, asthma affects all the airways, including the upper respiratory tract and nose.¹³ Airway inflammation in stable COPD is associated with the infiltration of primarily neutrophils, macrophages, and CD8⁺ lymphocytes that, in turn, mediate the stimulation and release of proinflammatory cytokines—leukotriene B₄, interleukin-8 (IL-8), and tumor necrosis factor- α (TNF- α).⁶ In addition, an imbalance of proteases and antiproteases and oxidative stress have been found to play a role in the inflammatory process in stable COPD.⁶ Airway inflammation in asthma, in contrast, is associated with the activation of primarily eosinophils and T_H2 CD4⁺ lymphocytes. In addition, mast cells and macrophages also play a role in the inflammatory cascade of asthma. CD4⁺ lymphocytes mediate the stimulation of the inflammatory cytokines IL-4, IL-5, and IL-13.¹³ It should be noted that although T_H2 CD4⁺ lymphocytes and mast cells are not an inflammatory component of stable COPD,¹⁵ during exacerbations of COPD, these cells can appear in the lungs of COPD patients.¹⁶ The clinical significance of this is discussed in *Management of Exacerbations*, page 8.

The different inflammatory processes in the 2 disease states result in different pathologic changes and responses to specific therapies that characterize each disease. In COPD, the pathologic

changes include squamous metaplasia of the epithelium of the proximal airway; chronic bronchitic changes, increased cholinergic tone, premature collapse of proximal airways during exhalation due to a loss of collagen and elastin to support the airway and keep it open; airway wall thickening, peribronchial fibrosis, luminal inflammatory exudates, and airway narrowing (obstructive bronchiolitis) of the peripheral airways; alveolar wall destruction and apoptosis of epithelial and endothelial cells of lung

parenchyma (emphysema); and thickening of intima, endothelial cell dysfunction, and remodeling of pulmonary vasculature.⁶ In asthma, characteristic pathologic changes include epithelial shedding, thickening of the basement membrane, mucous metaplasia, smooth muscle increases (hypertrophy and hyperplasia), and proliferation of blood vessels in airway walls.^{6,13} These pathophysiologic differences have important functional consequences and implications for therapy.¹⁷

Inflammatory and Pathophysiologic Differences Between COPD and Asthma

Category	COPD	Asthma
Cells	Neutrophils ++ Macrophages +++ CD8 ⁺ T cells	Eosinophils ++ Macrophages + CD4 ⁺ T cells
Key mediators	IL-8 TNF- α , IL-1 β , IL-6 NO +	Eotaxin IL-4, IL-5, IL-13 NO +++
Oxidative stress	+++	+
Site of disease	Peripheral airways (obstructive bronchitis) Proximal airways Lung parenchyma (emphysema) Pulmonary vessels	All airways
Pathologic changes	Squamous metaplasia Mucous metaplasia Small airway fibrosis Parenchymal destruction Pulmonary vascular remodeling	Fragile epithelium Mucous metaplasia Thickening of basement membrane Bronchoconstriction

NO = nitric oxide. Adapted from Doherty DE¹⁴; GOLD Guidelines 2009.⁶

TABLE 1. Clinical Differences Between COPD and Asthma

Characteristics	COPD	Asthma
Age at onset	Usually older, >40 years (but, depends on age of onset of smoking or environmental exposure)	Usually younger, often in childhood
Clinical features	Dyspnea: persistent or progressive Cough: chronic (often productive) Sputum	Dyspnea: variable Cough Wheeze: episodic with chest tightness
Etiology	Nonallergic Exposure to tobacco smoke or other environmental pollutants	Allergic; allergies present in >50% of patients Family history of asthma
Clinical course	Chronic, progressive	Usually intermittent
Airflow limitation	Partially reversible; after resolution of an acute exacerbation, may return to the level before the acute episode (irrespective of the baseline severity of the disease before the exacerbation); deteriorates with increasing age	Largely reversible after resolution of episode
Medical history	Allergies, sinusitis rare	Allergies, sinusitis Frequent respiratory infections (during childhood and adulthood) Nasal polyps
Smoking history	History of heavy smoking, >20 pack-years	Usually none
Common comorbid conditions	Smoking-related diseases: cardiovascular disease, osteoporosis, anxiety, depression	Atopic diseases: allergic rhinitis, allergic dermatitis
Treatment response		
Bronchodilators	Partially reversible airflow obstruction, decrease in air trapping/hyperinflation	Reversible airflow obstruction
Corticosteroids	Poor (little or no effect on inflammation)	Good (inhibits inflammation)

Adapted from Doherty DE¹⁴; Kuebler KK, et al.¹⁸

COPD patient as an elderly smoker is changing. Recent evidence indicates that COPD may occur at a much younger age after 10 to 20 pack-years of smoking.^{6,7} For example, if individuals start smoking at age 14, theoretically, they could have

an exposure significant enough to lead to the development of COPD in their mid-20s to early 30s. Asthma, in contrast, is associated most often with onset during infancy or childhood.¹¹ However, its clinical course can be bimodal; it can

disappear in adolescence and reappear in adulthood.¹⁴

Smoking

Smoking is a risk factor for the development of COPD and is a potential trigger leading to bronchospasm in asthma. In general, the risk for COPD increases with pack-years of smoking.^{6,11} Cigarette smokers with COPD have a higher prevalence of respiratory symptoms and lung function abnormalities, a greater annual rate of decline in lung function, and a greater COPD mortality rate than nonsmokers with COPD. The age at which an individual starts smoking, total pack-years smoked, and current smoking status are predictive of COPD mortality.⁶ Nonetheless, not all smokers develop clinically significant COPD, indicating that other factors (including genetic predisposition) are involved in determining susceptibility.²⁰ In asthma patients, smoking causes an increase in asthma symptoms and long-term impairment in lung function and reduces the efficacy of inhaled and systemic corticosteroids.¹³ Individuals with asthma who have never smoked also can develop fixed airway obstruction (airway remodeling).

Airway Hyper-responsiveness

The characteristic functional abnormality of asthma is airway hyper-responsiveness—an acute increased responsiveness to a stimulus that would be innocuous in a healthy person. The resulting airway narrowing leads to variable airflow limitation and intermittent symptoms.¹³ Airway hyper-responsiveness can be present in COPD; although its magnitude often is less than that occurring in asthma.¹⁴

Comorbidities

Asthma most often is associated with prior atopic (allergic) reactions and a family history of atopy or asthma.¹¹ Because

asthma may be triggered by allergies, increased levels of serum immunoglobulin E (IgE) commonly are found in asthma patients. Thus, eczema and rhinitis are common comorbid conditions in asthma patients.¹³ Interestingly, asthma also can occur in the absence of atopy (ie, normal serum levels of IgE), which is indistinguishable from atopic asthma in terms of inflammatory cell infiltrates and activated cytokine pathways in the pulmonary mucosa.^{21,22} In contrast, allergic etiologies are atypical in COPD.²³

COPD patients have a different comorbid profile from asthma patients. COPD patients are at increased risk for myocardial infarction, angina, osteoporosis, respiratory infection, bone fractures, anxiety, depression, diabetes, sleep disorders, anemia, and glaucoma.⁶ In addition, COPD is associated with extrapulmonary (systemic) effects including weight loss, nutritional abnormalities, and skeletal muscle dysfunction.

Clinical Course

The clinical course of COPD and asthma also differs. The clinical course of COPD is chronic, progressive with little day-to-day variation, while it usually is episodic in asthma.^{6,11} However, patients with COPD presenting with acute exacerbations of symptoms could be misdiagnosed as having recurring or poorly controlled asthma episodes. COPD exacerbations are triggered by upper respiratory infections and environmental factors such as air pollution, temperature changes, or exposure to tobacco smoke.⁶ Some patients with asthma with chronic mucus plug formation in the airways may not display the strong episodic course characteristic of the disease. Anti-inflammatory therapy in these patients may take weeks or months to clear the airway obstruction,¹³ resembling airway obstruction in COPD. A fundamental difference between COPD and asthma is that the airway

obstruction in COPD is not fully reversible with therapy,⁶ while it typically is fully or nearly fully reversible in patients with asthma.¹³

Despite the clinical differences between COPD and asthma, differential diagnosis may be complicated by the fact that 10% to 15% of patients with obstructive pulmonary disease have both COPD and asthma.²⁴ Some individuals with asthma who continue to smoke may later develop COPD in addition to their asthma.¹⁴ Moreover, patients (23%) with long-standing asthma (21–33 years) develop irreversible airway obstruction, termed airway remodeling, which is a characteristic feature of COPD.²⁵ In these patients, like in COPD patients, airflow obstruction will not be totally reversible (as measurable by postbronchodilator spirometry).

CLINICAL ASSESSMENT: Making the Diagnosis

When a patient presents with cough, dyspnea, and wheezing, symptoms common to both COPD and asthma, the clinical assessment should include a patient history, physical examination, and diagnostic testing for airflow limitation (spirometry).⁶

History and Physical Examination

Patient history can provide important clues for the differentiation between COPD and asthma. Patients should be asked specific questions regarding age at onset of symptoms, history of smoking, history of atopy, characteristics of cough (often dry in asthma; often productive in COPD), symptom variability (daily changes in symptom frequency or intensity are common in asthma; COPD symptoms are less variable), and family history.^{6,26} As patients with COPD often fail to acknowledge the symptoms associated with the disease early in its course, questions during history taking should be

individualized. For example, if patients are asked if they have dyspnea, most patients will likely respond in the negative. This is because patients with COPD slowly modify their lifestyle over years to decades to perform activities that do not result in dyspnea.¹⁴ Clinicians need to ask patients specifically about activities they did a few years ago and if shortness of breath now prevents them from doing these activities, instead of dispelling their inactivity level to older age, being out of shape, or having weight problems.

Physical examination rarely is diagnostic in COPD. Physical signs of airflow limitation (eg, relatively horizontal ribs, “barrel-shaped” chest, and protruding abdomen, reflecting the pulmonary hyperinflation seen in COPD) usually are not present until significant impairment of lung function has occurred.⁶

Spirometry

Spirometry is the most commonly performed noninvasive diagnostic test of lung function and is considered the best standardized, most reproducible, and most objective measurement of airflow limitation.⁶ Spirometry is considered the gold standard for establishing the diagnosis of COPD and monitoring its progression.⁶ However, spirometry alone is not sufficient to monitor disease progression or response to therapy; spirometry must be used in conjunction with patient history and current daily activity level.

Key spirometric measurements necessary for the early detection and monitoring of COPD and asthma are the total volume of air forcibly exhaled from the point of maximal inspiration (forced vital capacity [FVC]), volume of air exhaled during the first second of this maneuver (forced expiratory volume in 1 second [FEV₁]), and the calculated ratio of these 2 measurements (FEV₁/FVC).⁶ The peak expiratory flow (PEF) is an important measurement of lung function in asthma,

TABLE 2. Spirometric Differences Between COPD and Asthma

	FEV ₁	FEV ₁ /FVC
COPD^a disease severity		
Mild	≥80% predicted	<.7
Moderate	50%-79% predicted	<.7
Severe	30%-49% predicted	<.7
Very severe	<30% predicted or <50% predicted plus chronic respiratory failure	<.7
Asthma disease severity		
Intermittent	>80% predicted	Normal ^b
Mild persistent	>80% predicted	Normal ^b
Moderate persistent	60%-79% predicted	Reduced ≤5% from normal ^b
Severe persistent	<60% predicted	Reduced >5% from normal ^b

^aPostbronchodilator FEV₁ recommended for diagnosis and assessment.

^bNormal FEV₁/FVC = 85% for patients aged 8-19 years; 80% for patients aged 20-39 years; 75% for patients aged 40-59 years; 70% for patients aged 60-80 years. GOLD Guidelines 2009⁶; National Heart Lung and Blood Institute.¹¹

TABLE 3. COPD Management Goals

Component	Goal
Risk factor	Reduce exposure
Symptoms	Relieve
Pulmonary function/airflow limitation	Prevent progression of pulmonary dysfunction
Activity and exercise	Improve exercise tolerance (strength, endurance)
Exacerbations	Prevent and treat
Drug-related adverse event	Avoid
Disease-related mortality	Reduce

GOLD Guidelines 2009.⁶

but less so in COPD.¹⁴ Pre- and post-bronchodilator FEV₁ and FEV₁/FVC results may permit the differentiation of COPD from asthma, but also provide information to stratify the severity of the 2 diseases (Table 2).^{6,11} An important distinction between COPD and asthma is that airflow limitation (as measured by spirometry) is not fully reversible and does not return to normal in COPD patients even with therapy; in asthma patients, airflow limitation is largely reversible and should return to normal or near normal with appropriate treatment, the exception being patients with a component of fixed airflow obstruction due to airway remodeling.^{6,14}

Although spirometry is essential for evaluating airflow limitation, it requires effort to attain FVC and some patients have difficulty reaching FVC.²⁷ The measurement of FVC requires the patient to empty his/her lungs completely, a process that may take up to 20 seconds in individuals with severe obstruction; this can be physically exhausting for older or impaired individuals or those with severe respiratory problems. In addition, FVC is dependent on expiratory time, which varies with age in both normal individuals and those with airway obstruction, and there is no discrete end of test criterion. Consequently, the FEV₆ (FEV at 6 seconds of exhalation) has been adopted in office settings as a surrogate of the FVC and is endorsed by the National Lung Health Education Program.²⁸ FEV₆ has been validated and has the advantage that the end of a spirometric examination is more explicitly defined, easier to achieve, and less physically demanding for patients.^{27,29}

Other Diagnostic Tools

Chest x-ray and electrocardiography are not useful to diagnose early disease, but these tools may be useful late in the disease to identify comorbidities. However, in early disease, chest x-ray may be valuable

in excluding alternative diagnoses and establishing the presence of significant comorbidities such as cardiac failure and lung cancer. Radiologic changes associated with COPD (at later stages) include signs of hyperinflation (flattened diaphragm on lateral chest film and an increase in volume of the retrosternal air space), hyperlucency of the lungs, and rapid tapering of the vascular markings (suggestive of pulmonary hypertension). Electrocardiography may be of value in the diagnosis of cor pulmonale.⁶

MANAGEMENT OF COPD: What the Guidelines Say Goals of Management

Goals of COPD management are to reduce the progressive nature of the disease with a focus on reducing symptoms and exacerbations while improving exercise tolerance and quality of life (Table 3).⁶ Pharmacotherapy is central to preventing and reducing COPD symptoms and reducing the frequency and severity of exacerbations. None of the medications used in the management of COPD have been shown to significantly modify the long-term decline in lung function.⁶ As sustained smoking cessation potentially can slow further decline of airflow limitation, smoking cessation medications, at least indirectly, may delay the onset or slow the progression of COPD.^{30,31}

Due to the progressive nature of COPD, therapy typically involves a step-up approach whereby more treatments are added as disease worsens. Nonetheless, therapy should be individualized.⁶ Treatment regimens need to be patient-specific as the relationship between severity of symptoms and severity of airflow limitation is influenced by other factors, such as frequency and severity of exacerbations, presence of ≥ 1 complication, respiratory failure, comorbidities (eg, cardiovascular disease, sleep-related

disorders), and general health status. In addition, patient response to therapy is variable.⁶

Drug Therapy for Stable COPD

Several classes of drugs are available for the management of COPD (Table 4). Guidelines specify the order in which the drugs should be introduced into the treatment regimen. The order is based on disease severity and frequency of exacerbations.⁶ For mild disease, therapy is initiated with an as-needed short-acting bronchodilator. As disease progresses and symptoms become more constant

(moderate disease), maintenance bronchodilator therapy is started with a long-acting bronchodilator (eg, a long-acting anticholinergic or a long-acting β_2 -agonist). If symptoms persist, combination therapy is recommended whereby a second long-acting bronchodilator from a different class is added to the initial therapy. As disease progresses further (severe or very severe disease, FEV₁ <50% predicted) and is accompanied with frequent exacerbations, the addition of an inhaled corticosteroid to the combination long-acting bronchodilator therapy (an anticholinergic + a long-acting β_2 -agonist)

TABLE 4. Commonly Used Medications for the Management of COPD

Drug class	Example
β_2 -agonists	
Short-acting	Levalbuterol, albuterol, terbutaline
Long-acting	Formoterol, salmeterol
Anticholinergics	
Short-acting	Ipratropium bromide
Long-acting	Tiotropium
Combination short-acting β_2 -agonists plus anticholinergic in 1 inhaler	Albuterol/ipratropium
Methylxanthines	Aminophylline, theophylline (SR)
Inhaled glucocorticosteroids ^a	Beclomethasone, budesonide, fluticasone, triamcinolone, mometasone
Combination long-acting β_2 -agonists plus glucocorticosteroids in 1 inhaler	Formoterol (4.5 μ g)/budesonide (160 μ g) (only ^b), salmeterol (50 μ g)/fluticasone (250 μ g) (only ^c)
Systemic glucocorticosteroids	Prednisone, methylprednisolone

GOLD Guidelines 2009.⁶

^aMonotherapy with inhaled corticosteroids is not approved by the FDA for the treatment of COPD.

^bThis is the only dose combination of formoterol/budesonide approved by the FDA for treatment of COPD.

^cThis is the only dose combination of salmeterol/fluticasone approved by the FDA for treatment of COPD.

is recommended, assuming the patient has been adherent to all previously prescribed COPD medicines (Figure).⁶

It is important to note a fundamental difference in disease management between stable COPD and asthma: inhaled corticosteroids are used as second-line add-on therapy in COPD; but these agents are used as first-line therapy in asthma and are considered the mainstay of asthma therapy. Inhaled corticosteroids effectively reverse inflammation in asthma, but are less effective in controlling the chronic inflammation associated with stable COPD. This is because the primary inflammatory cells and inflammatory mediators in stable COPD (macrophages, CD8⁺ lymphocytes, and neutrophils) and asthma (mast cells, eosinophils, T_H2 lymphocytes) differ and while the former are

steroid nonresponsive, the latter are very steroid responsive.¹⁴

Chronic treatment with inhaled glucocorticosteroids is not recommended in stable COPD.^{32,33} However, inhaled corticosteroids may be used regularly in symptomatic COPD patients with an FEV₁ <50% predicted who have been maximally bronchodilated with an anticholinergic plus a β₂-agonist and were adherent to the bronchodilator regimen, but continue to have frequent exacerbations. When used in this setting, inhaled corticosteroids have been shown to further reduce the frequency of exacerbations beyond that achievable with the use of bronchodilators alone or in combination, and thus improve the health status of symptomatic COPD patients.³⁴

Management of Exacerbations

Exacerbations are acute in onset and represent a change in the patient's baseline dyspnea, cough, and/or sputum that is beyond normal day-to-day variations.⁶ Exacerbations are associated with a high mortality rate (≤49% 3 years after hospitalization for an exacerbation)³⁵ and negatively impact patients' quality of life³⁴ and lung function.³⁶

Management goals for exacerbations include prevention, early detection, and prompt treatment to minimize the risk of hospitalization, prolong the time to the next exacerbation, and improve quality of life.⁶ As most exacerbations are treated at home, patient education is important for the management of exacerbations as well as for prevention (see *Effective Patient Education Improves COPD Management*, page 9).⁷

		TREATMENT OPTIONS				
		Active reduction of risk factor(s); influenza vaccination	Add short-acting bronchodilator (when needed)	Add regular treatment with ≥1 long-acting bronchodilator (when needed); add rehabilitation	Add inhaled glucocorticosteroids if repeated exacerbations	Add long-term oxygen if chronic respiratory failure Consider surgical treatments
COPD STAGES	MILD FEV ₁ /FVC <.70 FEV ₁ ≥80% predicted	X	X			
	MODERATE FEV ₁ /FVC <.70 50% ≤ FEV ₁ <80% predicted	X	X	X		
	SEVERE FEV ₁ /FVC <.70 30% ≤ FEV ₁ <50% predicted	X	X	X	X	
	VERY SEVERE FEV ₁ /FVC <.70 FEV ₁ <30% predicted or FEV ₁ <50% predicted plus chronic respiratory failure	X	X	X	X	X

Figure. Treatment recommendations according to disease severity. GOLD Guidelines 2009.⁶

Inhaled bronchodilators, particularly short-acting inhaled β_2 -agonists (albuterol), with or without a short-acting anticholinergic (ipratropium), and short courses of systemic (oral or intravenous, if needed) corticosteroids, with or without a broad-spectrum antibiotic, are effective treatments for acute exacerbations of COPD.⁶ Evidence indicates that a short course (<14 days) of a systemic corticosteroid (prednisone) shortens recovery time, improves lung function (FEV₁) and hypoxemia, and prolongs the time to the next exacerbation (may reduce the risk of early relapse) or treatment failure, and shortens length of hospital stay in symptomatic patients experiencing exacerbations.^{37,38} During exacerbations, the cells and mediators that are in asthma (which are steroid-responsive) appear in the lungs of patients with COPD. This factor explains why systemic steroids are beneficial in COPD exacerbations, but not stable COPD. For the same reasons, inhaled corticosteroids are beneficial in patients with severe COPD who have frequent exacerbations (because asthma cells and mediators are frequently in the lungs of these patients).

Infection of the tracheobronchial tree and environmental exposures such as air pollution and primary- or second-hand smoke are the most common causes of exacerbation; although, the cause of about 30% of severe exacerbations are unknown.⁶ The consensus is that roughly 50% of severe exacerbations are due to noninfectious causes and approximately half of the remaining 50% are due to viral or bacterial infection.³⁹ Antibiotic treatment may, thus, be beneficial in patients experiencing exacerbations with clinical signs of airway infection such as increased sputum purulence and change in the color and/or consistency of sputum.

PCE TAKEAWAYS

- COPD often remains undiagnosed or is misdiagnosed as asthma and inappropriately treated.
- Airway obstruction in COPD is not fully reversible with therapy; in asthma, airway obstruction typically is fully or nearly fully reversible.
- Allergic etiologies are atypical in COPD; asthma often is associated with a history of atopic reactions.
- COPD has a chronic, progressive course, while asthma typically has an episodic course.
- Spirometry is considered the gold standard for establishing the diagnosis of COPD and monitoring disease progression.
- Drug therapy for COPD can include short- and long-acting bronchodilators and corticosteroids, but the specific regimen used must be individualized for each patient, based on disease severity and frequency of exacerbations.
- Patient education is key to preventing and managing COPD exacerbations.

Effective Patient Education Improves COPD Management

Patients need to be educated on how to recognize the symptoms of exacerbations. They also need to understand the importance of early treatment, the differences between maintenance medications (eg, inhaled long-acting bronchodilators and corticosteroids) and rescue medications (eg, short-acting bronchodilators), and proper use of systemic corticosteroids (prednisone) and antibiotics. Patients also need to be educated on the proper use of inhaled medications (bronchodilators and corticosteroids); training in inhaler technique is essential for all COPD patients. Patient education also should focus on basic and simple to understand information about COPD and the pathophysiology of the disease, smoking cessation, general approach to therapy and specific aspects of medical treatment, self-management skills, strategies to help minimize dyspnea, advice about when to seek help, and end-of-life issues.⁶ By understanding the nature of the disease, risk factors for progression, and their role and the role of the healthcare provider in achieving optimal management and health outcomes, patients will be better able to cope with living with COPD, more adherent to therapy, and improve their quality of life. The most important component of a successful interdisciplinary disease management approach to COPD (or any chronic disease) is to empower the patient to understand the disease process and the rationale for the medications prescribed.

CASE: 48-YEAR-OLD WOMAN WITH RECURRENT BRONCHITIS AND WHEEZING

Case Study With Commentary by Mary D. Knudtson, DNSc, NP, FAAN

A 48-year-old woman presents complaining of recurrent bronchitis and wheezing. She reports having a cold that developed about 10 days ago and has not resolved. She has a productive cough, with green sputum; shortness of breath; and wheezing.

History

The patient says she develops colds several times each year, which are usually treated with inhaled albuterol and oral antibiotics. She has no fever with these colds, and no other family members become ill around the same time she does.

She works full-time as a school teacher. She is married and has 2 children, 1 in high school and 1 in college. She smoked for 30 years, but quit 2 years ago after her father died of lung cancer. Her mother is alive at age 78. The mother has a history of hypertension and hyperlipidemia and was a smoker for many years, quitting when her spouse developed lung cancer a few years ago. The patient has 2 siblings, both alive and well—1 with hypertension and 1 with seasonal allergies.

The patient says she engages in no regular exercise, except cleaning house and running errands. She complains about becoming out of breath more easily as she has gotten older and thinks that she is just out of shape. Her diet includes coffee, juice, toast, and eggs in the morning; a hamburger, taco, or sandwich for lunch with soda; and a salad, vegetable, starch, and meat at dinner. She snacks on chips and ice cream.

Current Medications

Thyroid pill once daily; albuterol inhaler as needed. She asks for a refill of

her inhaler; she reports having no antibiotic allergies.

Physical Findings

- Height: 5 ft 4 in; weight, 182 lb; waist circumference, 35 in; temperature, 99.9°F; heart rate, 96 bpm; respiratory rate, 18 breaths per minute; blood pressure, 142/88 mm Hg
- Head: normocephalic/attraumatic
- Nares: clear, no erythema
- Pharynx: clear, no erythema, no exudate
- Nodes: slightly enlarged anterior cervical lymph nodes, nontender, mobile
- Ears: no erythema, decreased mobility of the tympanic membrane, serous fluid bilaterally
- Heart: normal S₁, S₂; apical pulse 94
- Lungs: diffuse wheezing throughout, no consolidation, + coarse rhonchi

Differential Diagnosis

Based on her presenting symptoms, the differential diagnosis includes COPD, asthma, acute exacerbation of chronic bronchitis, and viral infection.

The patient has a sibling with a history of allergy, which points to the possibility of asthma. However, her long-term exposure to cigarette smoke points to COPD as a possible diagnosis. To clarify the diagnosis, spirometry with and without

a bronchodilator is warranted. Some clinicians may want to consider a complete blood cell count and a chest x-ray as well.

Spirometry Findings

- FEV₁: 70% of predicted
- FEV₁/FVC: 60% of predicted
- <10% improvement postbronchodilator spirometry

Diagnosis

The <10% improvement in the post-bronchodilator spirometry is consistent with COPD, in which airway obstruction is not fully reversible with treatment. Her FEV₁ and FEV₁/FVC indicate moderate disease severity. The diagnosis is moderate COPD with an acute exacerbation of chronic bronchitis.

Treatment Decision

Regular use of a long-acting bronchodilator was prescribed. The acute exacerbation of chronic bronchitis was treated with oral antibiotics. She was advised to begin an exercise regimen as soon as the exacerbation resolved. The influenza vaccine and pneumococcal vaccines also were administered.

Commentary

It is very common for women with COPD to be undiagnosed. It is important for patients with risk factors for COPD and signs or symptoms of the disease to be given spirometry with and without bronchodilators to determine whether they have COPD, asthma, or another lung disorder. Early identification of patients with COPD allows appropriate management of the disease, patient education regarding smoking cessation, and preventive vaccine administration.

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