

A Breath of Life

Advances in Chronic Obstructive Pulmonary Disease

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The case of Nina

Nina, 64, presents to the clinic with a three- to four-year history of progressive, exertional shortness of breath and daily productive cough. Functionally, she is able to climb one flight of stairs and perform most of her usual daily activities. She has a history of smoking 45 pack-years and is currently considering quitting.

Results of the physical examinations performed are listed in Patient stats.

- What is this patient's diagnosis?
- Are there any interventions which will help to slow the progression of disease?
- What therapy, if any, should be started?
- Should this patient be considered for an exercise program?

For a followup on Nina, go to page 78.

In this article:

1. What is chronic obstructive pulmonary disease?
2. How is it diagnosed?
3. How can it be treated?

Patient stats

- Resting oxygen saturation: 94%
- Respiratory rate: 22/min
- Barrel chest appearance with percussion hyper-resonance and decreased breath sounds bilaterally
- No evidence of clubbing or congestive heart failure
- Hemoglobin: 162 g/L
- Chest X-ray: Hyper-inflation with upper lobe vascular marking deficiency
- Spirometry: Post-bronchodilator FEV₁: 1.1 L (38% predicted); FEV₁/FVC: of 56%
- Using salbutamol occasionally for relief

FEV: Forced expiratory volume; FVC: Forced vital capacity

What is COPD?

Chronic obstructive pulmonary disease (COPD) is a systemic disease largely caused by smoking. It is characterized by progressive,

partially reversible airflow obstruction, systemic manifestations, and increasing frequency and severity of exacerbations. Symptoms of shortness of breath and exercise limitation are often insidious at onset and are typically

progressive. The impairment in respiratory function and the systemic manifestations together contribute to reduced health-related quality of life, causing significant disability.

More than 500,000 Canadians over the age of 35 have self-reported symptoms of COPD. In 1998, COPD accounted for 4% of all deaths in Canada and was rated fourth as a cause of death for men and seventh for women. From 1988 to 1998 the mortality rate for women increased by 38%, while for men it decreased by 11%.¹

What are the risk factors?

The major risk factor for COPD is cigarette smoking, which accounts for 80% to 90% of cases.² Other environmental exposures linked to COPD include air pollutants (sulphur dioxide, particulates); occupational exposure to cadmium, silica dusts, and solvent fumes; and passive smoking. The most well-established genetic abnormality is alpha₁-antitrypsin (AAT) deficiency, which affects approximately 1 in 6,000 individuals.³

What are the predictors of increased mortality?

In a study that evaluated 1,016 patients admitted to hospital with COPD exacerbations, higher mortality was associated with certain predictors. These predictors were: low Acute Physiologic and Chronic Health Evaluation (APACHE) score; low body mass index; older

Table 1

Who should be screened for COPD?

- Symptomatic smokers/ex-smokers over 40
- Patients with chronic cough
- Patients who have sputum production
- Patients with increased exertional shortness of breath and/or long-lasting upper respiratory tract infections

age; worse functional status two weeks before admission; low partial pressure of oxygen in arterial blood (PaO₂)/fractional concentration of oxygen in inspired gas (FiO₂) ratio; history of heart failure; low albumin; and *cor pulmonale*. Overall, the 180-day mortality rate was 33%, while the two-year mortality rate was 49%.⁴ The following are predictors of decreased survival: a forced expiratory volume (FEV₁) < 30%; age over 65 years; low PaO₂.¹

How is COPD diagnosed?

The objective demonstration of airflow obstruction, by spirometry, is mandatory for the diagnosis of COPD. COPD diagnosis requires both a post-bronchodilator FEV₁ < 80% and an FEV₁/forced vital capacity (FVC) ratio < 70% predicted. A degree of reversibility may be present on spirometry and may fluctuate over time. However, if spirometry normalizes over time or with therapy, the diagnosis of COPD must be revoked and an alternate diagnosis considered (*i.e.*, asthma).¹

Spirometry screening should be undertaken in specific categories of patients (Table 1).¹

Screening for AAT deficiency is indicated in any COPD patient with atypical features, including

Table 2
Canadian Thoracic Society COPD classification

COPD stage	Symptoms
At risk	Asymptomatic smoker, ex-smoker or chronic cough/sputum, but post-bronchodilator FEV ₁ /FVC ≥ 0.7 and/or FEV ₁ ≥ 80% predicted (both required for COPD diagnosis)
Mild	Shortness of breath from COPD* when hurrying on the level or walking up a slight hill.
Moderate	Shortness of breath from COPD* causing the patient to stop walking after about 100 m (or after a few minutes) on the level.
Severe	Shortness of breath from COPD* resulting in the patient becoming too breathless to leave the house, or breathless after undressing; or the presence of chronic respiratory failure, or clinical signs of right heart failure.

* In the presence of non-COPD conditions that may cause shortness of breath (e.g., cardiac dysfunction, anemia, muscle weakness, metabolic disorders), patient symptoms may not appropriately reflect COPD disease severity. Classification of COPD severity should be undertaken with care in patients with comorbid diseases, or other possible contributors to shortness of breath.

FEV: Forced expiratory volume
FVC: Forced vital capacity
COPD: Chronic obstructive pulmonary disease

ria that avoid unknown FEV₁ thresholds in the COPD classification system, but emphasize the clinical significance of symptoms (Table 2).⁴

How can COPD be treated?

A multidisciplinary approach is important in the care of COPD patients (Figure 1). Health education can play a role in improving the ability to cope with the illness. Teaching of self-management skills, the benefits of proper nutrition, and the importance of exercise reconditioning are essential in improving the overall care of COPD patients. Education is also pivotal with smoking cessation.

Smoking cessation

Smoking cessation is proven to slow disease progression in COPD. In 2001, approximately 20% of Canadians over 15 still smoked on a regular basis.⁵ With smoking cessation, the decline in FEV₁ returns more towards that of a non-smoker and symptoms of cough, sputum, shortness of breath, and wheeze

early onset disease, positive family history, and those disabled by symptoms in their 40s or 50s.³

What do the Canadian Thoracic Society guidelines say?

Although there is epidemiologic evidence indicating that FEV₁ correlates with patient symptoms,¹ specific cutoffs have not been identified. The proposed Canadian Thoracic Society (CTS) guidelines have used previously validated crite-

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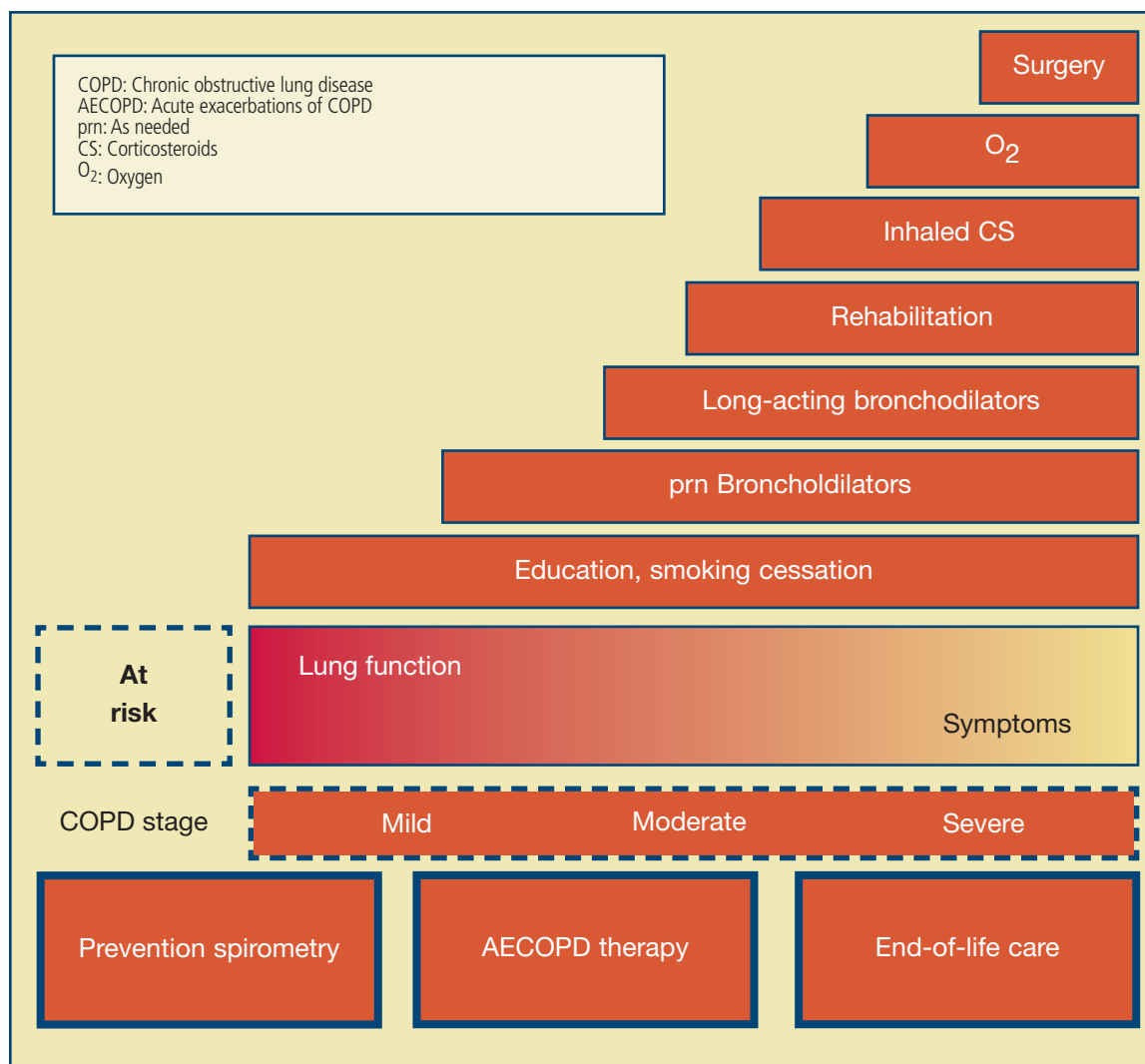


Figure 1. Individualized approach to COPD management.

improve. Additionally, the benefit of cardiovascular and cancer risk reduction should be emphasized.

Advice by physicians and other care-providers increases cessation rates. The use of smoking cessation aids, such as nicotine replacement and bupropion therapy, doubles the success rate.

Vaccination

All COPD patients should receive the influen-

za vaccine annually and the polyvalent pneumococcal vaccination every five years.

Pharmacologic therapy

The CTS guidelines recommend a stepwise, individualized approach, based on patient symptoms. Patients with symptoms occurring only with exertion and/or associated with mild disability should be started on a short-acting inhaled bronchodilator agent, such as salbuta-

A followup on Nina

The patient is told she has chronic obstructive pulmonary disease (COPD) and is advised and counselled to quit smoking. She is started on regular, daily tiotropium and told to use her short-acting beta agonist, salbutamol, as needed for shortness of breath. The benefits of the medication, appropriate use of the device, and potential adverse effects are reviewed. She is instructed to get an annual influenza vaccination and is referred to a pulmonary rehabilitation program.

A followup appointment is scheduled for two weeks to assess her response to therapy and the outcome of her efforts to quit smoking.

mol, ipratropium bromide, or combivent, two to four puffs, as needed.

If the patient experiences persistent symptoms, and/or is using short-acting bronchodilator therapy frequently, the addition of long-acting bronchodilators is recommended. Long-acting bronchodilators are more convenient to use and have proven benefits over short-acting bronchodilators when used as maintenance therapy. Options include the recently released long-acting anticholinergic agent, tiotropium, or long-acting beta agonists, such as salmeterol or formoterol.

If symptoms persist, a combination of tiotropium and long-acting beta agonists is recommended, although there are no published trials to support this recommendation yet. Short-acting beta agonists should continue to be used as needed at this stage.

In more severe disease, or in patients with frequent exacerbations, combination therapy with an inhaled corticosteroid/long-acting beta agonists preparation and tiotropium should be considered.

For persistent, severe symptoms, long-acting oral theophylline can be considered, although the prescribing physician must be wary of side-effects and potential drug interactions. Regular monitoring of drug levels is necessary.

Pulmonary rehabilitation

Formal pulmonary rehabilitation includes a multidisciplinary team whose focus is on the care of patients with chronic respiratory disease. This team provides an individually designed program to optimize social and physical performance. Patients who are symptomatic despite bronchodilator therapy

should be considered for pulmonary rehabilitation. In general, benefit can be obtained in patients with COPD regardless of the severity of impairment, age, gender, or nutritional status.

Treatment of exacerbations

The long-lasting detrimental effect of COPD exacerbations is now becoming better understood, and the short-term mortality associated with exacerbations is 10% to 20%.⁶ The diagnosis of COPD exacerbation is made with at least two of the following three findings:

- worsening dyspnea,
- an increase in sputum purulence, and
- increase in sputum volume.

Initial investigation with chest X-ray is recommended in most cases, as changes in management are made in up to 23% of cases based on chest X-ray findings alone.^{7,8} Routine spirometry is not recommended. Aggressive short-acting bronchodilator therapy should be added to the patient's usual regimen. If

patients are already taking tiotropium, adding therapy with regular short-acting beta agonists (as needed) is recommended.

In moderate to severe exacerbations, systemic corticosteroids (*i.e.*, Solu-Medrol[®], then prednisone for a total of two weeks) have been shown to be effective in reducing treatment failure and short-term re-exacerbation rates. In less significant exacerbations, oral prednisone (30 mg/day to 50 mg/day) should be prescribed for seven to 14 days. Non-invasive positive pressure ventilation should be considered for patients with moderate respiratory acidosis in the absence of contraindications. The cautious use of supplemental oxygen (to keep oxygen saturation at 90% to 92%) is advocated.

Patients fulfilling the definition of an exacerbation and who have sputum purulence should be treated with antibiotic therapy. A second- or third-generation cephalosporin with a macrolide; amoxicillin/clavulin; or a respiratory fluoroquinolone should be considered.

Surgery

Surgical options, such as bullectomy and lung volume reduction surgery, remain controversial and available to only a limited number of patients. Lung transplantation should be considered in patients with an FEV₁ < 25% and clinically significant hypoxemia, hypercapnia, pulmonary hypertension, rapid decline in lung function, or frequent severe exacerbations.

Oxygen therapy

All patients with severe COPD should be evaluated for home oxygen therapy. Previous

trials demonstrated that long-term administration of supplemental oxygen for over 15 hours per day, to keep oxygen saturation > 90% in patients with COPD, increased survival and improved quality of life.^{9,10}

Referral

Referral to a respirologist should be considered for patients with severe COPD, patients who fail to respond to appropriate therapy, patients severely disabled, and patients with multiple co-morbidities.

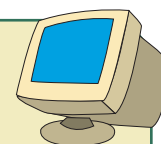
End-of-life issues

The quality of life for patients with advanced COPD is often poor. Physicians providing care for patients with COPD are strongly encouraged to undertake honest, informative, and empathetic discussions with patients regarding disease prognosis, patient concerns, and end-of-life issues.

The major issues affecting quality of life in end-stage COPD include dyspnea, cough, retained secretions, depression, and disrupted sleep. Palliation of dyspnea and cough in the terminal stages of COPD is appropriate and is achieved with opiates and/or low-dose benzodiazepines. Generally, respiratory depression is an uncommon problem if patients are initi-

Web sites

1. The Canadian Lung Association:
www.lung.ca
2. COPD Today Online Magazine
www.healthyresources.com/copd






Take-home message

Diagnosis

- The diagnosis of COPD requires both a post-bronchodilator $FEV_1 < 80\%$ and an FEV_1/FVC ratio $< 70\%$ predicted.
- Spirometry screening should be undertaken in symptomatic individuals.

Treatment

- Smoking cessation is proven to slow disease progression in COPD.
- Both short-acting and long-acting bronchodilators can lessen symptom severity.
- Exacerbation of symptoms can be treated with corticosteroids.
- All patients with severe COPD should be evaluated for home oxygen therapy.
- Treatment is effective and can provide symptomatic relief.

ated on a low dose and titrated slowly. Retained secretions may be responsive to systemic anticholinergic agents, such as a scopolamine patch applied every 72 hours. It is appropriate to use a multidisciplinary palliative care team to optimize the care provided to patients nearing the end of life. 

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