



COPD: More options mean potentially better control

Some common diseases outside of my specialty areas of interest have, over time, blended into the background of daily practice. When patients have clearly inadequately controlled disease I refer them back to their internist or relevant subspecialist. Otherwise I find myself accepting the clinical status quo, providing stopgap care when necessary, utilizing my present knowledge of the disease process. But if I am not aware of any paradigm shifts in management, I tend to just peruse the published studies of new drug therapies as they appear in major journals, and not necessarily incorporate new therapies into my practice.

Chronic obstructive pulmonary disease (COPD) is one of those diseases, and the update by Hatipoğlu and Aboussouan in this issue of the Journal (page 373) provided the opportunity to review some important things I have missed and to take note of the therapeutic principles that haven't changed that much.

The incidence of COPD seems to be increasing. Cigarette smoking remains the major reversible cause, but one-fourth of current COPD patients have never smoked.

Patients who have early COPD may not be asked about or may not volunteer complaints specific to COPD. Lung examinations are insensitive, and chest radiography, if done, rarely identifies patients with early disease, so it is no surprise that COPD is underdiagnosed. While for many patients it is unclear whether the natural progression of lung damage can be dramatically altered by early diagnosis, other than by stopping smoking, that may not be the case for subsets of patients such as those with alpha-1 antitrypsin deficiency. But COPD generally has to be suspected in order to prompt appropriate screening for it.

COPD is still diagnosed by spirometry, with findings demonstrating incompletely reversible airway obstruction. The forced expiratory volume in 1 second (FEV_1), a spirometric measure required to make the diagnosis, has been a useful surrogate marker of drug efficacy, but it is far from the ideal measure of disease progression and risk of death. Patients who have frequent exacerbations requiring hospitalization tend to have a tumultuous clinical course. The best predictor of future exacerbations is the patient's history of exacerbations. Reducing the exacerbation rate improves the quality of life but may not translate to a benefit in terms of the mortality risk.

A lower risk of death is most consistently attained by helping patients stop smoking and, if they have sustained hypoxemia, giving them supplemental oxygen. The former intervention is the population game-changer—yet the US Centers for Disease Control and Prevention reports that 18% of American adults still smoke cigarettes, a number that dropped only slightly between 2005 and 2012. Americans with less education and less income more frequently smoke cigarettes, and it is estimated that one in 13 Americans currently 17 years or younger will die prematurely from the effects of cigarette smoking.

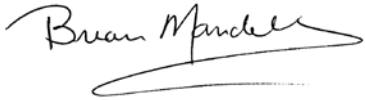
As for newer therapies, many that are applicable to most patients with COPD represent a fine-tuning of drug therapies we have been using for decades. Thus, it

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is not surprising that no dramatic increase in survival rates has been achieved. The advantage of several of the newer therapies in terms of FEV₁ or exacerbation rate is statistically significant but relatively modest in terms of clinical impact. Their clinical promise (including specific antibiotics in low doses), revealed in recent studies, may be dependent upon using them in the most appropriate patients. Reducing the exacerbation rate is a significant advantage, but I would also like to know which drugs or drug combinations are most likely to permit major reductions in patients' corticosteroid requirements. Hopefully, these data will come from real-world comparative efficacy studies.

Reading the article by Hatipoğlu and Aboussouan, I was reminded that utilizing our pulmonary colleagues' expertise is of particular value in individualizing therapy for our patients with COPD—using newer drugs and combinations when most appropriate, not just because they are readily available. As physicians who are not pulmonary specialists, we need to be vigilant in recognizing the treatable comorbidities that contribute to dyspnea and poor outcome in patients with COPD, such as obstructive sleep apnea, congestive heart failure, obesity, and thromboembolism.

But to make the largest impact on the mortality rate, we need to continue to engage in and expand the ongoing war against cigarette use.



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