Is Hospice Referral Ever Appropriate in COPD?

The author was recently asked by a hospice organization, of which he is a board member, to write an article for the group’s bulletin concerning appropriate referral of patients with COPD for hospice care. The first reaction was that this idea is intrinsically a “non-starter.” By regulation, hospice referrals are supposed to be for patients in whom “significantly increased mortality within six to twelve months can be expected,” though progenes in cases of COPD are known to be highly variable. However, a recently published set of standards for diagnosis and care of COPD indicated that patients generally have a poor outlook if they have “poor baseline function, marginal nutritional status, severely restricted activity, and inexorable deterioration of [their remaining lung function]. . . .”

On further reflection, and after discussion with colleagues who are knowledgeable about issues of medical ethics, an idea emerged that may be worth proposing to the pulmonary medicine community. Would it not be appropriate, in some situations, to propose referral to a hospice organization if, at the time of one’s discussion with a COPD patient about intubation and mechanical ventilation, the patient declines to consider aggressive intervention? This is not the place to detail if, but hospice care has a number of advantages over routine, unorganized, palliative-type care.

The proposal, then, would be to consider referral of patients with severe COPD who decline intubation and mechanical ventilation to a local hospice group. The National Hospice Organization has listed proposed guidelines for use in establishing severity of disease, which must of necessity be tentative. A slightly modified and shortened version of these criteria is presented below.

I. Severity of chronic lung disease documented by:
A. Disabling dyspnea at rest, poor response to bronchodilators resulting in decreased functional ability (eg, bed-to-chair existence), often exacerbated by other debilitating symptoms such as fatigue and cough. FEV₁ after bronchodilator is helpful supplemental objective evidence, but should not be required if not already available.
B. Progressive pulmonary disease, with increasing visits to an emergency department or hospitalizations for pulmonary infections and/or respiratory failure. Decrease in FEV₁ of greater than 40 mL per year on serial testing is helpful supplemental objective evidence, but should not be required if not already available.

II. Presence of cor pulmonale or right-heart failure. These should be due to advanced pulmonary disease, not primarily or secondary to left-heart disease or valvulopathy. These may be documented by methods as follows: physical signs of right-heart failure on echocardiography, ECG, or chest radiograph (these studies or findings should not be required for certification for the Hospice Benefit). The “Hospice Benefit,” is “available as a benefit under Medicare Hospital Insurance (Part A) to beneficiaries with a very limited life expectancy. A Medicare beneficiary who chooses hospice care receives non-curative medical and support services for his or her terminal illness. Home care is provided along with necessary inpatient care and a variety of services not otherwise covered by Medicare.”

III. Hypoxemia at rest. Either Po₂ ≤ 55 mm Hg or oxygen saturation ≤ 88%.
IV. Hypercapnia, with Pco₂ ≥ 50 mm Hg.
V. Unintentional progressive weight loss of greater than 10% of body weight over the preceding 6 months.
VI. Resting tachycardia greater than 100 beats/min in a patient with known severe COPD.¹

Henry Yeager, Jr., MD, FCCP
Washington, DC

REFERENCES

Hypertension and Heart Failure
The Link Continues

When Franklin Roosevelt entered his fourth term as President of the United States in 1944, he was dying of congestive heart failure (CHF). He had severe uncontrolled hypertension, and no therapy was available to significantly modify its course. The Framingham study began in 1948, 3 years after Roosevelt’s death from a cerebral hemorrhage. Early data from that study demonstrated that hypertension was the major risk factor in the development of CHF.¹ Since then, randomized clinical trials in hypertensive patients have demonstrated that treatment results in a marked reduction in the development of heart failure.²-⁵ With wide availability of effective, well-tolerated antihypertensive therapy, many believed that identification and treatment of hypertension had reduced, if not nearly eliminated, it as a major risk factor for the development of CHF. The focus of cardiology for the last 10 years has been on the treatment of established CHF and on therapeutic approaches to prevent the progression from asymptomatic left ventricular dysfunction to overt heart failure. The Survival of Left Ventricular Dysfunction (SOLVD) trials⁶-⁷ and the Survival and Ventricular Enlargement (SAVE) trials⁸ have clearly shown the efficacy of angiotensin-converting enzyme (ACE) inhibitor therapy in patients with left ventricular systolic dysfunction. Many have viewed most modern-day left ventricular dysfunction as the long-term price we pay for reducing the mortality from acute myocardial infarction.

The assumption that hypertension is no longer an important risk factor for the development of CHF was clearly dispelled this past year by Levy and colleagues⁹ from Framingham, Mass. These investigators evaluated the role of hypertension as a risk factor for CHF in the “modern era.” Using a start date of January 1, 1970 (a time when good antihypertensive therapy was widely available), 5,143 subjects (age 40 to 89) from the original Framingham cohort, and subjects from the Framingham Offspring Study were enrolled. All subjects were free of CHF as determined by the standard Framingham major and minor criteria system. Followed for an average of 14.1 years, 392 new cases of CHF were identified in this cohort. Hypertension predated the onset of CHF in 91% of these cases. Hypertension increased the hazard ratio for the development of CHF in men to 2.07 and in women to 3.35. After adjusting for other risk factors (coronary artery disease, diabetes mellitus, left ventricular hypertrophy, and valvular heart diseases), hypertension carried the greatest population-attributable risk for the development of CHF of all the risk factors considered. Hypertension was the attributable risk for the development of CHF in 39% of men and 59% of women. While higher blood pressure carried greater risk for CHF, even subjects with stage 1 hypertension had a substantially increased risk of developing CHF.

While it’s no great surprise to any of us in the heart failure field, it is also important to note that the survival associated with the development of overt heart failure was dismal. In the Levy et al Framingham study,⁹ the hypertensive men who developed heart failure had a median survival of only 1.37 years, and in women the median survival was 2.48 years. At 5 years after identification of overt heart failure 76% of the men and 69% of the women had died.

Why does hypertension remain a risk factor for CHF?²

We know from a number of large treatment studies (Veteran’s Administration Cooperative Study Group on Antihypertensive Agents,²-³ Systolic Hypertension in the Elderly Program [SHEP],⁴ and Swedish Trial in