On Depression, Antidepressant Medications, and Resuscitation Preferences in COPD Patients

To the Editor:

In their excellent article (January 2005), Dr. Stapleton and colleagues described the association of high depressive symptoms on the Center for Epidemiologic Studies-Depression (CES-D) scale with a lower preference for cardiopulmonary resuscitation (CPR) in patients with COPD. Approximately 36% of their study subjects reported a history of clinical depression as a current coexisting illness. It is not clear why the authors did not examine the association between end-of-life preferences and the presence of self-reported depression given that clinical depression was already diagnosed; the presence of high depressive symptoms measured by the CES-D indicates possible depression. I thus wonder if the authors would have had similar results if self-reported clinical depression was used as a predictor of treatment preferences for CPR and mechanical ventilation. Reanalyzing their data using self-reported depression may also help in explaining the discrepancy in their finding of a lower preference for CPR and a similar preference for mechanical ventilation by subjects with CES-D scores ≥ 16 when compared to those with scores CES-D < 16.

In their discussion, the authors rightly suggest that patients responding to antidepressant medications might change their end-of-life preferences as their moods improved. In view of this suggestion, it would also be important if the authors could analyze their data examining whether the preference rates for CPR among the depressed COPD patients might change with adjustment for antidepressant medication use in the multivariate analyses. Adjustment for antidepressant medication use might lend further support to the need for end-of-life preferences reassessment after an adequate trial of antidepressants. In addition to improving mood, antidepressant medications may have additional benefits for other common COPD comorbidities: reduction of tobacco craving, palliation of subjective dyspnea, improvement of appetite with weight loss reversal, and lowering of anxiety symptoms. The nihilistic attitude fostered by depressive symptoms and other common psychological comorbidities in COPD patients may dissipate with antidepressant use, possibly leading to a more informed decision regarding end-of-life care preferences. Given the high prevalence of depression in the COPD population, screening for (and early treatment of) depression in these patients should be part of routine care, as treatment might improve their overall quality and quantity of life. A large, controlled trial of the impact of antidepressants on overall well-being and survival of COPD patients with depression is long overdue.

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Mechanism of Mucin Secretion in Diffuse Panbronchiolitis

To the Editor:

We read with interest the article by Kim et al¹ in CHEST (September 2004) in which the authors examined the relationship between epidermal growth factor receptor (EGFR) expression in the bronchial epithelium with neutrophilic inflammation and mucus hypersecretion in the tissues of patients with diffuse panbronchiolitis (DPB). DPB is a COPD that is characterized clinically by chronic cough, excessive sputum, and dyspnea. It is prevalent in Japan and Korea but is rare in other countries. If left untreated, DPB is fatal. The introduction of low-dose, long-term macrolide therapy in 1984 caused the survival rate to rise markedly.² Since then, most studies had concentrated on the mechanisms while very few had been looking for the mechanisms of the disease itself. Therefore, the effort of Kim et al¹ to investigate the mechanism of mucus hypersecretion in DPB and its relation to the EGFR-mucin pathway should be acknowledged. However, several points of weakness in their study should be debated.

First, it is known that in healthy lungs few cells will be positively stained with alcian blue/periodic acid-Schiff (AB/PAS) stains. The markedly high percentage of mucin staining in the bronchial epithelium of the control samples in this study (50%) indicate clearly that there was something wrong with these samples. The authors mention that the samples were taken from healthy portions of the lobectomy specimens from six nonsmoking patients with adenocarcinoma. It seems that even the uninvaded lung tissues of the adenocarcinoma patients were showing secondary changes, so clearly these patients were not the proper control subjects. The authors could use the easier option of collecting fiberoptic bronchoscopic bronchial biopsy specimens from completely healthy volunteers.

Second, Figure 7 seems to contradict what is written in the results and is shown in Figure 4. While in the results it is written that the mean (± SD) percentage of the luminal area occupied by AB/PAS and MUC5AC stains was 84.6 ± 7.63% in the DPB group, which is beautifully shown in Figure 4; Figure 7 shows hardly any intraluminal mucin or MUC5AC staining in either group.

Third, in the control subjects, the finding that 50% of the bronchiolar epithelial area was occupied by epithelium stained with AB/PAS and MUC5AC while EGFR expression was absent is difficult to understand. It had been shown that the airway mucin production in response to various stimuli is mediated through the EGFR cascade.³ EGFR was shown to be related to mucus production in goblet cells, not to the degranulation of the mucin granules.⁴ The authors’ assumption that goblet cell hyperplasia occurred due to some transient inflammation, and that degranulation did not occur due to inflammation subsidence, which led to a down-regulation of EGFR, is too hypothetical, and it only indicates that these were not suitable control samples.

In conclusion, we think that another, better controlled prospective study that will also examine the BAL fluid for counts of neutrophils and other cells, as well as for cytokines such as interleukin-8, and their correlation to the EGFR expression and mucus secretion is still needed.

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Plasma Levels of N-Terminal Pro-Brain Natriuretic Peptide in the Critically Ill

The Right Hormonal Marker in the Wrong Patients?

To the Editor:

A recent article by Jefic and coworkers¹ (July 2005) on the utility of N-terminal pro-brain natriuretic peptide (NTproBNP) for estimation of pulmonary artery occlusion pressure (PAOP) in critically ill patients concluded that NTproBNP may be a strong discriminator of cardiac dysfunction in these patients. The authors observed inverse correlations between NTproBNP and cardiac index and left ventricular stroke work index (LVSWI) but not between NTproBNP and PAOP. This suggests that, in contrast to patients with heart failure,² other factors than the physiologic stimulus ventricular stretch may be involved: the accompanying disease process (sepsis,³ surgery⁴) and pharmacologic factors.

We have shown that 15 mL/kg of NaCl 0.9% IV induces a 250% increase in NTproBNP levels in volunteers.⁵ The patients of Jefic et al¹ will have been treated with a much higher volume/sodium load and, additionally, many drugs that have not been studied yet regarding effects on NTproBNP. Thus, the correlations between NTproBNP, cardiac index, and LVSWI may be an epiphenomenon of underlying disease and therapy, and increased NTproBNP levels in these patients should better be interpreted as signs of multiorgan dysfunction instead of cardiac dysfunction.

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