Brain Natriuretic Peptide Measurement in Patients With COPD and Cardiovascular Disease

To the Editor:

We read with great interest the recent article by Maclay and MacNee in CHEST (March 2013). The authors discussed different potential mechanisms that link COPD to an increased risk of cardiovascular disease, which will be the focus of novel therapeutic targets in the near future. We feel, however, that the relation among brain natriuretic peptide (BNP), COPD, and cardiovascular disease must be addressed.

It is well established that COPD is associated with increased cardiovascular mortality and ventricular dysfunction. In a 2011 prospective cohort study, the prevalence of ventricular dysfunction among patients with COPD was 17%, with an increased risk of mortality during the 2-year follow-up period (hazard ratio, 2.34; P = .053). BNP has been proposed as a noninvasive marker to detect previously unknown left ventricular dysfunction in patients with acute exacerbation of COPD. A 2010 prospective study in a cohort of patients with acute exacerbation of COPD found a strong correlation between levels of N-terminal pro-BNP and early mortality. In addition, BNP is an independent predictor of endothelial dysfunction. Therefore, using BNP as a surrogate marker in patients with COPD may help identify patients with underlying cardiovascular disease. The physician has to keep in mind that the natriuretic peptide level elevation in this setting is much more discrete that the one found in overt left ventricular failure.

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Response

To the Editor:

We thank Drs Kheir and Salerno for their interest in our review. We agree that measuring N-terminal B-type natriuretic peptide (NT-proBNP) levels in COPD is of interest. Chang et al reported that in patients admitted with exacerbation of COPD, both plasma NT-proBNP and troponin T levels predicted mortality at 30 days, even after accounting for other markers of exacerbation severity. While these markers seem to reflect myocardial stretch and myocardial ischemia during acute exacerbation of COPD, it is less likely that NT-proBNP and troponin T are mediators of cardiovascular disease in COPD. In addition, it would be difficult to ascertain whether NT-proBNP release in exacerbation of COPD is due to right-sided heart dysfunction rather than left-sided heart dysfunction.

Drs Kheir and Salerno comment that BNP is an independent predictor of endothelial dysfunction. BNP was shown to be an independent predictor of acetylcholine-mediated vasodilatation, a sensitive measure of systemic endothelial function. We have previously compared endothelial function using this gold standard method and showed no differences between patients with COPD and control subjects matched for age and cigarette smoke exposure.

Thus, in summary, measuring NT-proBNP may have clinical relevance in exacerbation of COPD and may reflect myocardial strain in this circumstance. However, its contribution is likely to be as a marker rather than a mediator of cardiovascular dysfunction in COPD.

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References