Response

To the Editor:

I thank Lippi and Targher for their kind remarks about my editorial in CHEST (February 2008),1 which describes “tricks of the trade” for assessing pulmonary embolism prognosis. They raise two important questions: (1) Should measurement of natriuretic peptides such as brain natriuretic peptide or pro-brain natriuretic peptide be incorporated into routine prognostic assessment of acute pulmonary embolism? (2) Do brain natriuretic peptide levels provide clinically useful, complementary information above and beyond troponin measurement?

Troponin measurement has several advantages over natriuretic peptides. First, any elevation of troponin, even a “troponin leak,” carries a markedly increased adverse prognosis for survival over the ensuing 30 days. Second, troponin as a pulmonary embolism biomarker has been far more extensively studied than natriuretic peptides. Third, the normal range for natriuretic peptides and the level at which they indicate a poor prognosis for pulmonary embolism remain uncertain. Fourth, the negative predictive value for a normal troponin level is almost 100%. Therefore, it is hard to conceive how natriuretic peptides can yield incrementally useful clinical information to indicate a favorable prognosis. Fifth, dissecting the contribution of occult or overt concomitant heart failure to elevated natriuretic peptide levels in the presence of pulmonary embolism is difficult.

For now, we know that normal troponin levels indicate a likely excellent prognosis. I hope that some day soon, we will be able to use the combination of elevated troponin levels plus elevated natriuretic peptide levels as an indicator of an especially ominous prognosis. However, that day has not yet arrived. Therefore, routine measurement of natriuretic peptides is not quite ready for prime time when assessing the prognosis of a patient with acute pulmonary embolism.

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REFERENCE


Transient Ventricular Dysfunction Syndrome

The Evolving Nomenclature

To the Editor:

I would like to congratulate Kurowski and colleagues on their excellent work on tako-tsubo cardiomyopathy and the proposal of “Transient Left Ventricular Dysfunction Syndrome.” The description of this novel syndrome has suffered with the lack of a consistent nomenclature and its ever-evolving picture through various reports, including the present one. For example, many case reports and case series on right ventricular involvement with a similar regional distribution as the left side have been published.2–5 Elesber et al3 were the first to describe it on a large scale. In their study published on 25 patients with transient left ventricular apical ballooning syndrome, 8 were found to have significant right ventricular apical involvement on echocardiography and it was associated with a longer and critical hospitalization. Haghi et al4 reported 9 of 34 patients (26%) with tako-tsubo cardiomyopathy with regional motion abnormalities in right ventricle (visualized on cardiac MRI). In eight of nine such patients, these segmental abnormalities cleared up with follow-up. Of note, the patients also had high incidence of certain underlying conditions (systemic hypertension, hypercholesterolemia, diabetes, Graves’ disease, COPD and paroxysmal atrial fibrillation) which have the potential to adversely affect global right ventricular function. Yet, the finding seems significantly important with abnormalities being more regional and transient in nature. The distinction may be important for at least two reasons. First, the disorder affects more “globally” than it was earlier thought and second, right-sided involvement signifies a more severe impairment in left ventricular systolic function and poorer short-term prognosis as suggested by these reports. To conclude, it appears that “Transient Ventricular Dysfunction Syndrome” may thus be a more accurate choice instead of “Transient Left Ventricular Dysfunction Syndrome” in the face of existing evidence.

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