Commentary

B-type natriuretic levels in critically ill patients: critically misleading?

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Abstract

Although B-type natriuretic peptide (BNP) has been used for the diagnosis of congestive heart failure in many clinical settings, its diagnostic role in critically ill patients remains uncertain. The body of literature suggests that BNP and N-terminal pro-BNP levels are not useful for the diagnosis of systolic or diastolic heart failure in the critically ill, including in patients with brain hemorrhage, due to poor specificity. However, these cardiac peptides may have a more promising prognostic role in this patient population.

In the previous issue of *Critical Care*, Meaudre and colleagues measured bedside rapid assay B-type natriuretic peptide (BNP) levels daily, and performed bedside echocardiography in patients admitted to hospital for subarachnoid hemorrhage (SAH) [1]. They found that BNP levels rose in 25/31 (81%) patients, peaking at day 2 (at a mean of 126 ng/ml) and tapering off by day 7. Importantly, BNP levels did not correlate with left ventricular (LV) filling pressure as estimated by echocardiography. BNP levels did correlate with cardiac troponin I levels at day 2 and day 3 ($R = 0.63$, $P < 0.001$ and $R = 0.44$, $P = 0.05$, respectively). The authors therefore concluded that BNP cannot estimate LV filling pressure in SAH patients but does correlate with myocardial necrosis as assessed by cardiac troponin I levels in these patients without prior hypertensive or cardiac disease.

What is the mechanism of BNP release in such patients? As Meaudre and colleagues mention, intramyocardial norepinephrine release, possibly resulting in myocardial necrosis, appears to be a plausible mechanism of BNP release – and perhaps explains the correlation of BNP with troponin I levels [1,2]. It should be noted that BNP levels, putatively correlating with intramyocardial norepinephrine levels, do not necessarily correlate with serum norepinephrine levels, which makes sense given the lack of clinically detectible myocardial injury (lack of decrease in LV ejection fraction, lack of new wall motion abnormalities) in these patients [1,2].

There are several important caveats – many of which the authors mention – that must be considered when interpreting Meaudre and colleagues' data. First, all patients with known hypertensive or cardiomyopathic disease were excluded from the study. The extent of neurocardiogenic injury associated with SAH in patients with known cardiovascular disease is therefore unknown and could arguably be more serious. Second, although echocardiographic diagnosis of the LV filling pressure has significant precedent and is clinically useful [3], the diagnosis remains inferior to direct hemodynamic data from right or left heart catheterization, which was not performed in this study. Third, and importantly, the 6/37 (16%) patients with SAH who died prior to the day 7 follow-up studies were excluded from the final analysis, and consequently it is unknown whether BNP or troponin I levels had any prognostic or mechanistic effect in these expired patients.

What is the significance of these findings? These data add to the growing body of evidence that BNP (and N-terminal pro-BNP) are poor predictors of LV filling pressure in critically ill patients. In critical care patients with indwelling pulmonary artery catheters, it has previously been demonstrated that BNP levels have a weak correlation ($R = 0.32$) with the pulmonary capillary wedge pressure [3]. In another study of 40 critical care patients, both BNP ($R = 0.40$) and N-terminal pro-BNP ($R = 0.32$) had weak correlations with the pulmonary capillary wedge pressure and were dependent on renal function [4]. Another group has shown that, in 249 critically ill patients, those with congestive heart failure confirmed by invasive hemodynamic measurements had BNP and N-terminal pro-BNP levels comparable with patients with sepsis and without congestive heart failure [5]. Yet another

BNP = B-type natriuretic peptide; LV = left ventricular; SAH = subarachnoid hemorrhage.

study showed that BNP levels were increased in critically ill patients with severe sepsis or septic shock regardless of the presence or absence of heart failure [6]. The body of evidence therefore indicates that BNP and N-terminal pro-BNP should not be used to diagnose systolic or diastolic heart failure in critically ill patients.

Is this the death knell, then, for the use of BNP in critical care? From a diagnostic point of view, yes – BNP and N-terminal pro-BNP levels do not provide useful, cardiac-specific information given that sepsis, trauma or congestive heart failure can result in similar BNP levels in critically ill patients [6-8]. From the prognostic point of view, however, the role for cardiac peptides could be more promising. While one study in patients with sepsis showed that BNP levels did not predict inhospital mortality or length of stay [6], two other studies in unselected critically ill patients showed that hospital nonsurvivors had significantly higher NT-pro-BNP values than hospital survivors [7,8]. A potentially important prognostic role therefore remains for cardiac peptides in critically ill patients, an area that requires further study; however, the diagnostic role of BNP and N-terminal pro-BNP in such patients appears to be very limited indeed.

Competing interests

The author declares that they have no competing interests.

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