Usefulness of B-type Natriuretic Peptide Levels in Predicting Hemodynamic and Clinical Decompensation

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KEYWORDS

• BNP • Pulmonary congestion
• Heart failure • Decompensation

There are more than 3 million heart failure (HF)-related admissions annually in the United States, and approximately 35% of these admitted patients subsequently have HF-related deaths or readmissions within 60 days.[1–3] Improving methods of assessing preclinical decompensation should be important in reducing HF-related morbidity and mortality.

Natriuretic peptide (NP) levels (ie, B-type natriuretic peptide [BNP] and *N*-terminal pro BNP [NT-proBNP]) have become a mainstay in the diagnosis or exclusion of acute HF.[4] Emerging evidence suggests that NP levels also might be valuable adjuncts in both treatment monitoring and assessment for possible clinical decompensation.[5–9] Patients who have chronic HF often are on a tenuous portion of the pressure–volume curve, and small volume shifts can mean the difference between pulmonary edema and renal failure. These subtleties often are difficult to discern with clinical examination and chest radiographs. Because BNP usually correlates with pulmonary capillary wedge pressure (PCWP),[10] it should be useful for detecting early subclinical congestion, and this identification can help preventing acute clinical decompensation.

DECOMPENSATION IS RELATED TO PULMONARY CONGESTION

With the increasing use of implantable defibrillators and beta-blockers, pulmonary congestion, rather than low cardiac output, is becoming the leading cause of hospital admissions and death in patients who have HF. In the Vasodilatation in the Management of Acute Congestive Heart Failure trial, the mean wedge pressure of patients admitted for HF was high (25–30 mm Hg), and they tended to have a preserved cardiac index.[11] Similarly, in the Acute Decompensated Heart Failure National Registry of more than 100,000 patients, only 3% of patients admitted with decompensated HF had evidence of low cardiac output with a systolic blood pressure lower than 90 mm Hg.[12]

Previous studies also have shown that high PCWP at discharge is a poor prognostic indicator. In the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness trial, PCWP was an important predictor of 6-month postdischarge survival.[13] In an observational study of patients hospitalized for decompensated HF, a reduction of PCWP to below 16 mm Hg at discharge was associated with decreased 2-year mortality, whereas a cardiac index above 2.6 L/min/m^2 was not associated with improved outcome (**Fig. 1**).[14] Thus the wedge pressure is an important diagnostic and therapeutic target.

The cascade that ultimately leads to pulmonary congestion often begins with an increased blood

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volume leading to increased left ventricular end-diastolic pressure and elevated PCWP. High pressures reflected into the lungs lead to redistribution in the pulmonary vascular bed with subsequent interstitial edema followed by alveolar edema. This pulmonary congestion is manifested clinically by symptoms of shortness of breath and orthopnea and by lung rales on examination. Eventually, increased pulmonary artery pressure may lead to elevated right ventricular and right atrial pressures. This increased pressure causes clinical symptoms of systemic, or right-sided, congestion such as jugular venous distension and peripheral edema (**Fig. 2**). As shown in **Fig. 3**, in a study of 32 patients who had chronic HF implanted with a hemodynamic monitor, there was a rise in measures of volume overload up to 7 days before hospitalization for acute decompensated HF.[15] Thus there is an important preclinical window in which congestion is masked; if congestion is detected and addressed during this window, hospitalizations may decrease (see **Fig. 3**).

Congestion secondary to an increase in left ventricular wall stress (especially diastolic) also is accompanied by rapid synthesis and release of BNP from the cardiac myocyte.[16] Resolution of clinical congestion occurs before the restoration of normal cardiovascular hemodynamics. One reason patients who clinically seem to be euvolemic on discharge decompensate after discharge may be that their subclinical hemodynamic derangements are still present.[3]

CONGESTION IS DIFFICULT TO ASCERTAIN ON PHYSICAL EXAMINATION

Even in the best of hands, establishing the diagnosis of HF may be clinically challenging. Physical examination findings such as rales, increased jugular venous pressure, and edema are specific but not sensitive for elevated cardiac filling pressures.[17] In one recent study, the third heart sound was 92% specific for HF but was only 41% sensitive.[18] Also, in a subset of patients who have HF, there is a discrepancy between jugular venous pressure and PCWP. Some patients may have a predominantly right-sided HF with pulmonary hypertension and may have elevated jugular venous pressure but a normal lung examination. Conversely, patients who have systolic HF but intact right heart function may have high wedge pressure and low central venous pressure. In addition, radiographic findings often lag behind clinical findings and thus are not always useful in diagnosing acute decompensation or clinical improvement.[19] Thus, hemodynamic abnormalities such as elevated PCWP do not always correlate with clinical symptoms and signs and may go unrecognized until patients present with decompensation. The lack of sensitivity of the clinical findings in establishing the diagnosis of pulmonary congestion also may make it difficult to ascertain the point in the clinical course of treatment when euvolemia has been established.

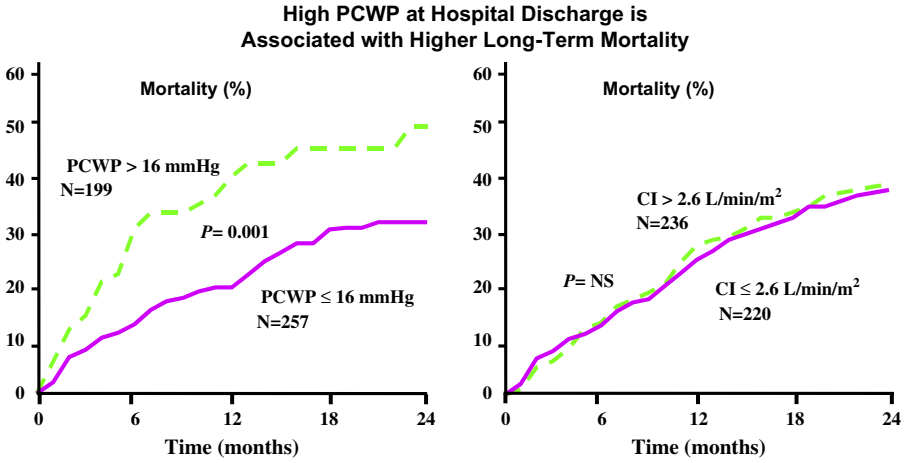

Fig. 1. In an observational study of patients hospitalized for decompensated HF, reduction of PCWP to less than 16 mm Hg was associated with reduced mortality, whereas the cardiac index was not associated with mortality in this patient group. CI, cardiac index. (*Data from* Fonarow GC, Stevenson LW, Steimle AE, et al. Persistently high left ventricular filling pressures predict mortality despite angiotensin converting enzyme inhibition in advanced heart failure. Circulation 1994;90:1–488; with permission. *Courtesy of* M. Gheorgiade, MD, Chicago, IL.)