LETTER TO THE EDITOR

Caveats in the interpretation of natriuretic peptide levels

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I read the article entitled ''B type natriuretic peptide as a predictor of anterior wall location in patients with non-ST-elevation myocardial infarction'' by Ramos et al.[1] in the March 2011 issue of Clinics with great interest. Although the finding is instructive, I think it would be more informative if several factors were also considered:

First, renal function was not evaluated in Ramos et al. 's population. B-type natriuretic peptide (BNP) is a small polypeptide with a plasma half life of 20–30 minutes that reflects the stress sustained by the left ventricle (LV) and that correlates well with the LV filling pressure and the heart failure status.[2,3] Several important studies have elucidated the fact that this miraculous myocardial stress marker can be affected by chronic kidney disease (CKD). In the PRIDE (PRoBNP Investigation of Dyspnea in the Emergency department) study, Anwaruddin et al. discovered that the N-terminal pro-BNP (NT-proBNP) level is inversely associated with the glomerular filtration rate (GFR) and that a higher cut-off value should be set for patients with poorer renal function (GFR$<$60 ml/min).[4] Takami et al. also tested this theory in a group of CKD patients who had not undergone dialysis and found that the BNP level was significantly increased compared with hypertensive patients with normal renal function.[5] They also concluded that LV overload (higher end-diastolic volume and pressure) may correlate fairly well with the BNP level, independent of the severity of renal dysfunction. McCullough et al. provided further convincing evidence of the graded relationship between BNP cut-off values and the GFR, further supporting the view that the interpretation of BNP levels should build upon the differential range of renal function.[6] Theories concerning the elevation of BNP in CKD patients include poor tolerance of fluid loading and resultant subclinical volume overload in early CKD stages, a higher degree of LV hypertrophy and dysfunction in advanced CKD stages, and decreased clearance of biomarkers as renal function deteriorates.[6,7] Given this understanding, it is prudent to include renal function, specifically creatinine data, in the assessment of the impact and predictive power of the BNP level.

In addition, age by itself may also influence the BNP level. Fabbian et al. recently performed an elegant study to investigate the confounding factors in decoding the NT-proBNP level in elderly patients. They discovered that in a medium-sized geriatric population, with a mean age of 80 years old, elevated NT-proBNP levels were associated with lower hemoglobin levels, more atrial fibrillation and less severe pulmonary disease and were significantly influenced by the CKD stage.[8] These results are indicative of the more complex situation that we might face with the elderly population, which is representing an increasing proportion of our society as time passes. In conclusion, considering the various obstacles that may hinder the elucidation of the relationship between the BNP level and patient-level outcomes, it is of vital importance that we gather other relevant information needed to determine the truth.

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