



Letter to the Editor

Natriuretic peptide levels in constrictive pericarditis

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Over and above achievements as biomarkers of heart failure [1], natriuretic peptides also help to differentiate constrictive pericarditis (CP) from its close mimic, congestive heart failure (CHF) attributable to restrictive cardiomyopathy (RCM), apparently irrespective of the New York Heart Association (NYHA) functional class [2,3].

In a comparison between 29 CP patients with mean NYHA functional class 2.65 vs 20 RCM subjects with mean NYHA functional class 2.7, NT-pro BNP levels were significantly ($p = 0.0001$) lower in CP than in RCM [2]. In a study which compared 6 CP patients vs 5 RCM patients, all eleven patients being in NYHA functional class III or IV, mean plasma Brain Natriuretic Peptide (BNP) levels were significantly ($p < 0.001$)

lower in CP subjects than in RCM subjects [3]. Myocardial stretch is the predominant mechanism for BNP release. Accordingly, CP-related impairment of BNP release might be attributable to the fact that “myocardial stretch is prevented by the constraining pericardium” [3]. Given the fact that, in general, natriuretic peptide levels are either normal or only modestly elevated in CP [2,3], there should be a high index of suspicion for CP when such levels are encountered in a patient with a provisional diagnosis of CHF, especially when other stigmata of CP coexist, such as markedly elevated jugular venous pressure [4], which remains persistently elevated despite diuretic therapy [5].

Over and above the few available studies on CP-related natriuretic peptide secretion, more studies are needed to improve the diagnostic utility of BNP in CP.

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