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COMPARISON OF BRAIN NATRIURETIC PEPTIDE LEVELS IN DECOMPENSATED CONGESTIVE HEART FAILURE DUE TO VARIOUS CAUSES

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Contribution
All the authors contributed significantly to the research that resulted in the submitted manuscript.

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ABSTRACT

Introduction: TThe high levels of brain natriuretic peptide (BNP) in patients with non Q MI, rheumatic heart disease, restrictive cardiomyopathy and lone atrial fibrillation indicate that mechanisms beyond stretch and dilatation of the heart may be operational in the secretion of BNP from the myocardium.

Objectives: To compare the blood BNP levels in patients with decompensated CHF due to rheumatic heart disease (RHD), chronic obstructive pulmonary disease (COPD) or non-Ischemic cardiomyopathies with the BNP levels in patients with decompensated CHF due to coronary artery disease (CAD).

Methodology: We enrolled 91 consecutive patients admitted as decompensated CHF due to CAD, RHD, COPD or non ischemic cardiomyopathy. Blood samples for BNP assay were taken. Data analysis was done using student's t-test and coefficient of simple correlation using SPSS-10 software.

Results: Out of 91 CCF patients (54 male), 51 had CAD, 17 RHD, 05 COPD and 18 had non ischemic cardiomyopathy. Compared to blood BNP levels in CAD (2081±1434 pg/ml) there was no significant difference in the blood BNP levels in RHD (1811±1137pg/ml, p=0.484), COPD (2355±1595 pg/ml, p=0.688) and non ischemic cardiomypathies (1951±1322 pg/ml, p=0.737).

Conclusion: Compared with the blood BNP levels in decompesated CHF due to CAD, the levels in RHD, COPD and non-Ischemic cardiomypathies were not significantly different.

Key Words: Natriuretic peptide, Heart failure, Echocardiography.

INTRODUCTION

The gratifying reduction in the mortality due to acute myocardial infarction and arrhythmias has resulted in an increase in the elderly population with congestive heart failure (CHF) due to coronary artery disease (CAD)[1]. The tremendous burden of CHF on the resources necessitates finding the ways to prevent its occurrence, to halt its progression and to minimize the sufferings from it[2]. In the diagnosis of CHF brain natriuretic peptide (BNP) has been shown to supercede the symptoms and physical signs including phonocardiographic S^3 and S^{43},[3,4]. Moreover the diagnostic and prognostic role of BNP has been well validated[5,6]. The predominant mechanism of BNP secretion from the heart is ventricular overload7. The most prevalent cause of CHF across the globe is coronary artery disease and hypertension8. Rheumatic heart disease (RHD) relatively rare in the west, is the second most common cause of CHF in Indo-Pak subcontinent[9,10], while chronic obstructive pulmonary disease (COPD) has been reported to be the third common cause[11].

OBJECTIVES: To compare the blood levels of BNP in patients presenting with decompensated CHF in NYHA class III or IV dyspnea due to rheumatic heart disease, cor pulmonale or cardiomyopathies with the BNP levels in patient presenting with decompensated CHF in NYHA class III or IV dyspnea due to coronary artery disease.

Hypotheses: The rise in BNP level in congestive heart failure is influenced by the etiology of CHF.

METHODOLOGY

This cross-sectional study was conducted in Cardiology department -from 15th December 2005 to 7th June 2006), during this period total of 91 patients with symptoms and signs of heart failure with various causes were recruited. Patients with decompensated CHF in NYHA class III or IV dyspnea for whom hospitalization was mandatory for stabilization and Consent for recruitment into the study were included in the study.

Patients with Congenital heart disease, Acquired ventricular septal defect, pericardial diseases and renal failure having serum creatinin more than 2 mg/dl were excluded from the study.

Decompensated CHF was defined as patient admitted with NYHA class III or IV dyspnea, raised JVP, edema and S3,NYHA class III dyspnea was defined as dyspnea on less than ordinary activity i.e. on getting out of bed to the bedside toilet,NYHA class IV dyspnea was defined as dyspnea at rest i.e. in the bed with head end elevated, CAD was diagnosed on history or evidence of ischemia on ECG, exercise test, coronary angiography or cardiac imaging, RHD was defined as having deformed regurgitant or stenotic valves on echocardiography, COPD was defined as right ventricular hypertrophy and dilatation caused by diseases of lung parenchyma and/or pulmonary vasculature unrelated to left side of the heart while Cardiomyopathies defined as Causes of congestive heart failure with systolic or diastolic dysfunction other than CAD, RHD and COPD.Blood BNP levels were determined by micro particle enzyme immunoassay (MEIA) technique using AXSYM (Abbott) BNP assay. AXSYM (Abbott) BNP assay is in harmony with the FDA approved Biosite triage Point of care BNP assay having more than 90% concordance with it12. The data was collected according to the proforma and entered into a notebook with duplicate pages. The data was also saved in the computer in Excel and SPSS-10 and written on non rewritable compact disc to guard against any computer crash.

Statistical analysis: The data were fed to a computer and analyzed using SPSS version-10 software. The mean and standard deviation of blood levels of BNP in decompensated CHF were determined for each etiological group. Blood BNP level in RHD, COPD and cardiomyopathy were compared with the BNP levels in coronary artery disease using independent sample t-test and ANOVA with a p value of less than 0.05 taken as significant. Box plots were used to show the difference in the BNP levels among these four groups. Independent sample t-test and box plot was used to find the difference between the BNP levels in patients with NYHA class III and IV. Independent sample t-test and box plot was also used to find the difference between the BNP levels in patients with and without atrial fibrillation.

RESULTS

Out of 91 CCF patients 54 male (Table-01). Out of 51 had CAD, 17 RHD, 05 COPD and 18 had non ischemic cardiomyopathy (Figure 1).

Thirty three patients had NYHA class III dyspnea while 58 had NYHA class IV dyspnea. Sixty four patients were in sinus rhythm while 27 had atrial fibrillation.

Compared to blood BNP levels in CAD (2081±1434 pg/ml) there was no significant difference in the blood BNP levels in RHD (1811±1137pg/ml, p=0.513, , COPD (2355±1595 pg/ml, p=0.688, pg#32) and non ischemic cardiomypathies (1951±1322 pg/ml, p=0.737. Using one way ANOVA the difference between the BNP levels in the four etiologic groups was not significant (p=0.840).

There was significant difference in the BNP levels in the NYHA III and IV (884±685pg/ml vs. 2666±1207pg/ml, p=0.000, (Figure 03).

There was no significant difference in the blood BNP levels in

Table 1: Frequency Based on Sex

Variables	Frequency	Percent
Male	54	59.3
Female	37	40.7
Total	91	100.0

Figure 1: Bar Diagram showing frequency of diagnostic / etiologic groups

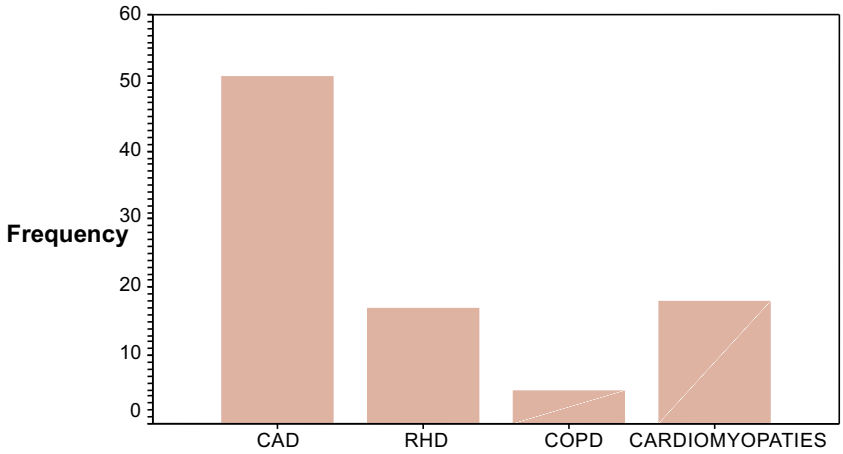


Figure 2: Box plot showing BNP levels in the four etiologic groups

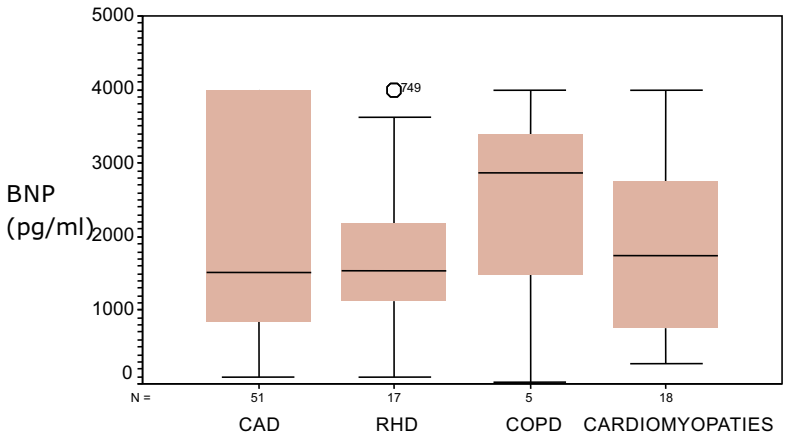
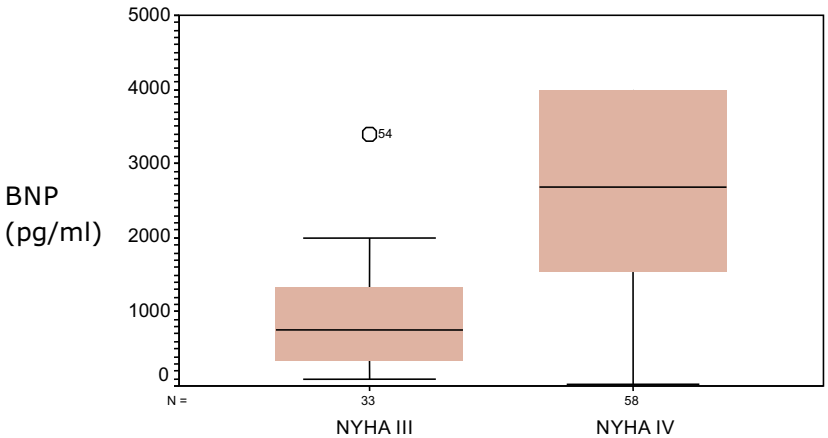


Figure 3: Box plot showing BNP levels in NYHA class III and IV



those with and without atrial fibrillation (2058±1330 vs. 1928±1428.

DISCUSSION

The number of COPD patients was small because many patient having COPD with or without CHF were dealt with by pulmonology unit and did not meet our inclusion criteria. More over the number of patients in the four etiologic groups does not represent true point prevalence. We stopped recruiting patients with CAD when their number reached fifty one. In our study there was no significant difference in the blood BNP levels in patients with CHF due to CAD, RHD, COPD and cardiomyopathies. Although we did not find any study to compare our result with. The subtle changes in the BNP levels due to pathologic process were probably overshadowed by the predominating bulk release due to ventricular overload. In our study the blood BNP levels in patients having NYHA class IV were significantly higher than from those having NYHA III dyspnea. Our results tally with the results of Wieczorek SJ et al where the BNP levels increased with the severity of CHF based on NYHA class[13]. BNP can thus be used as an objective measure for dyspnea and an alternative to six minute walk test to assess the severity of heart failure[14]. In a study Lee SC et al found that BNP levels correlated best with the NYHA functional class both at baseline and follow up so much so that they concluded with remarks that plasma BNP is a useful objective biomarker in monitoring human CHF in the outpatient setting[15]. In our study there was no significant difference in the blood BNP levels in those with and without atrial fibrillation, although elevated level of plasma BNP level have been reported in patients with AF in the absence of heart diseases[16].

Our null hypothesis was accepted i.e. the difference in BNP levels in CHF due to CAD, RHD, COPD and cardiomyopathies was not statistically significant.

The secondary finding which came out very strongly was the very significant difference in BNP levels based on the NYHA functional class. This fact, if validated by larger randomized studies will go a long way towards the objective assessment of functional status in CHF.

STUDY LIMITATIONS

1. Small sample size

2. Uneven etiologic groups

3. Difference based on NYHA class was not included in our objectives

CONCLUSION

1. Compared with the blood BNP levels in decompensated CHF due to CAD, the BNP levels in RHD, COPD and non-Ischemic cardiomyopathies were not significantly different.

2. BNP level was significantly higher in patient with NYHA IV than NYHA III dyspnea.

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