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ORIGINAL ARTICLE

Prognostic value of brain natriuretic peptide in acute pulmonary embolism

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KEYWORDS

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Abstract *Background:* Plasma levels of brain natriuretic peptide (BNP) correlate closely with the pressure and mural tension of the right ventricle and with pulmonary arterial pressure. The relationship between brain natriuretic peptide increase in acute pulmonary embolism (APE) and the increase in mortality and morbidity has frequently been suggested in small studies but its global prognostic performance remains largely undefined.

Methods: BNP levels were determined among the 30 consecutive patients arriving at the emergency department with shortness of breath and moderate to high probability of pulmonary embolism. All patients were subjected to a thorough clinical and physical examination, 12 lead electrocardiography, chest X-ray, echocardiography, laboratory tests including complete blood count, coagulation profile, renal and liver functions, brain natriuretic peptide and multiline computed tomography.

Results: 20 patients were diagnosed with APE. Among them, 7 were diagnosed with massive embolism (group 1) and 13 had mild to moderate embolism (group 2). Among the first group 6 patients died while only 2 patients from the second group died. BNP levels were significantly higher in group 1 (130 ± 15 pg/ml) compared to group 2 (91 ± 7 pg/ml) ($P < 0.05$) and this was associated also with an increased mortality in group 1.

Conclusion: BNP levels can help predict the severity and possible outcome in patients with pulmonary embolism.

Introduction

Accurate risk stratification in patients with pulmonary embolism (PE) is of first importance in selecting the optimal management strategy for each individual and to potentially improve patient outcome [1]. Indeed, in-hospital mortality associated with PE depends on clinical features at admission and increases significantly when the right ventricular (RV) dysfunction is documented by echocardiography even in the

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absence of hemodynamic deterioration [2]. Brain natriuretic peptide (BNP) is a neurohormone secreted from cardiac ventricles in response to ventricular strain [3]. It has been suggested that BNP might be valuable biomarkers for the diagnosis of the RV dysfunction in acute PE and subsequently to predict mortality and serious adverse events (SAEs), especially in patients with initial normal hemodynamic status [4].

Methods

This study included 30 patients arriving at the emergency room with complaints of shortness of breath and moderate to high probability of pulmonary embolism. All patients had a full medical and physical examination followed by multislice (16) computed tomography to confirm the diagnosis. Other investigations included electrocardiography, chest X-ray, echocardiography and laboratory tests including full blood count, coagulation profile, renal and liver functions and brain natriuretic peptide assay. According to the results of multislice CT, 10 patients had no evidence of pulmonary embolism and were used as the control group of the study group (3). The remaining 20 patients were divided into 2 groups: group 1 including 7 patients with massive pulmonary embolism and group 2 included the remaining 13 patients with mild to moderate pulmonary embolism.

For BNP assays we used a quantitative fluorescence immunoassay by Biosite (San Diego, USA) with a cutoff value of 90 pg/ml.

All patients who were eligible for thrombolytic therapy were given t-PA. There were initially infused with 20 mg loading bolus followed by 80 mg infusion over 2 h. This was followed with full dose enoxaparin 1 mg/kg every 12 h. Patients with mild and moderate severity embolism who were hemodynamically stable and had no RV dilation or hypokinesia were kept on full dose enoxaparin 1 mg/kg every 12 h.

Statistics

All results were tabulated and results were generated accordingly.

Statistical significance was reached when $P < 0.05$.

Results

30 patients were included in this study, divided to 3 groups based on multislice CT findings into: group 1 including 7 patients with massive PE; group 2 including 13 patients with mild to moderate PE, and group 3 including 10 patients with normal spiral CT representing the control.

All patients were subjected to complete history taking and physical examination. Baseline characteristics are shown in Table 1.

Systolic blood pressure was significantly lower in group 1 compared to the other groups, while the heart rate was significantly higher in groups 1 and 2 compared to group 3. Right ventricular S3 gallop was equally heard in groups 1 and 2 and this was statistically significant when compared to group 3.

Regarding ECG changes, more patients in group 1 (86%) had RBBB compared to group 2 (31%) and only 20% in group 3. This was statistically significant.

The biochemical profile including kidney and liver functions, coagulation profile, D-Dimer and BNP were done for all patients. All results are shown in Table 2.

The only statistically significant differences between the 3 groups were seen with the D-Dimer and BNP levels which were statistically significant for group 1 when compared to both groups 2 and 3. BNP Levels were also statistically higher when group 2 was compared to group 3.

Echocardiography with Doppler study was done for all patients. All patients in group 1 had right ventricular (RV) dilatation and hypokinesia when compared to group 3. 46% of patients in group 2 had also RV dilation and 31% had RV hypokinesia. RV systolic pressure was significantly higher in group 1 while left ventricular ejection fraction (LVEF) was significantly lower in group 1 largely due to paradoxical septal motion resulting from RV pressure overload. Data are shown in Table 3.

6 patients (86%) in group 1 died while only 3 patients (23%) in group 2 died. Mortality was zero in group 3. Major bleeding occurred more with thrombolytic therapy in group 1 while minor bleeding was more in group 2 with low molecular weight heparin. These data are shown in Table 4.

Table 1 Baseline characteristics of the studied groups.

Variable		Group 1 $n = 7$	Group 2 $n = 13$	Group 3 $n = 10$	$P < 0.05$
Age (Mean ± SD)		51 ± 10	58 + 8	55 ± 12	
Sex	Male	2	5	5	
	Female	5	8	5	
SBP		88 ± 9	102 ± 10	110 ± 4	$P < 0.05$
HR		114 ± 12	110 ± 11	90 ± 11	$P < 0.05$
RV S3		7	6	5	$P < 0.05$
ECG					
	RBBB	6	4	2	$P < 0.05$
	AF	2	1	0	
	S1, Q3, T3	1	10	8	

SBP = systolic blood pressure; HR = heart rate; RV S3 = right ventricular 3rd sound; ECG = electrocardiogram; RBBB = right bundle branch block; AF = atrial fibrillation.