Journal of Critical Care 33 (2016) 262–265

Contents lists available at ScienceDirect

Journal of Critical Care

journal homepage: www.jccjournal.org

Rapid Communication

A single BNP measurement in acute heart failure does not reflect the degree of congestion☆,☆☆

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ARTICLE INFO

Keywords:
Heart failure
B-type natriuretic peptide

ABSTRACT

Introduction: Multiple studies found a significant correlation between B-type natriuretic peptide (BNP) level and clinical severity of heart failure (HF). We aim to study the ability of a single BNP measurement to predict the degree of congestion in acute systolic HF.
Methods: Patients enrolled in the ESCAPE trial who were admitted with acute systolic HF were divided into tertiles according to baseline BNP level with comparison of the degree of congestion across tertiles using clinical signs of congestion as well as objective parameters of overload checked by the pulmonary artery catheter.
Results: A total of 251 cases (mean age, 56 years; 75% males) were included in the study after excluding patients with normal (n = 43) or extremely elevated BNP (n = 53) due to the known limited significance of BNP in predicting the degree of congestion in the latter 2 instances. These cases were divided into tertiles as follows: tertile 1, BNP less than or equal to 376 pg/mL; tertile 2, BNP 377 to 792 pg/mL; and tertile 3, BNP greater than or equal to 793 pg/mL. There were significant differences across the BNP tertiles in age (P = .03) and body mass index (P = .003). There were no differences between the 3 BNP tertiles with regard to the presence of rales (P = .533), jugular venous distension (P = .245), positive hepatojugular reflux (P = .224), hepatomegaly (P = .489), ascitis (P = .886), lower extremity edema (P = .068), or S3 gallop (P = .512). With regard to hemodynamic markers of congestion measured via the pulmonary artery catheter, there were no significant differences across the BNP tertiles in the right atrial pressure (P = .148), pulmonary capillary wedge pressure (P = .140), pulmonary artery systolic pressure (P = .155), pulmonary artery diastolic pressure (P = .246), and pulmonary artery mean pressure (P = .607).
Conclusion: Although longitudinal BNP follow-up may be valuable in reflecting the degree of congestion, looking at a single BNP measurement alone is not a good marker to predict the level of congestion and should not be used as a "stand-alone" test for determining aggressiveness of diuresis. Management should be guided by the entirety of physical examination, laboratory values, and hemodynamic parameters when available.

1. Introduction

Patients hospitalized with heart failure (HF) suffer high mortality and readmission rate despite continued advancements in medical treatment and device therapy [1]. Available data show that the main reason for hospitalization for HF are symptoms of congestion rather than low cardiac output. Grading congestion in acute HF is achieved through a combination of bedside assessment, laboratory markers and central hemodynamic variables of congestion if available. The B-type natriuretic peptide (BNP) is secreted in the cardiac ventricles following volume and pressure overload [2] and has aided in the diagnosis of HF in patients with elevated levels who are presenting with dyspnea. Multiple studies have found a significant correlation between BNP and clinical severity of HF [3]. Nonetheless, we have previously reported that a normal BNP in patients admitted with acute systolic HF has limited diagnostic ability due to similarities in the degree of congestion compared with those with elevated BNP and the fact that it may be a indication of the short BNP half-life of 23 minutes where 2-hours are need to reflect changes due to acute HF [4,5]. We have also found that an extremely elevated BNP in patients hospitalized with acute systolic HF has an equally limited diagnostic ability when utilizing a single admission measurement to predict the degree of congestion which was comparable in those with or without extremely elevated levels. Herein, we aim

☆ There are no conflicts of interest.
☆☆ No funding has been received for this manuscript.
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http://dx.doi.org/10.1016/j.jcrc.2016.02.023
0883-9441/

Table 1
Demographics, clinical, laboratory, hemodynamic and echocardiographic characteristics of patients enrolled in the ESCAPE trial admitted with acute systolic heart failure compared according to BNP tertiles.

	Tertile 1 BNP ≤ 376 (n = 84)	Tertile 2 BNP 377–792 (n = 83)	Tertile 3 BNP ≥ 793 (n = 84)	P-value
Baseline demographics				
Age (years, m ± SD)	52.9 ± 13.6	58.5 ± 12.9	56.2 ± 14.1	0.03
Male sex % (n)	58 (69%)	63 (75.9%)	66 (78.6%)	0.344
White race % (n)	48 (57.1%)	57 (68.7%)	54 (64.3%)	0.295
Black race % (n)	23 (27.4%)	16 (19.3%)	22 (26.2%)	0.420
BMI on admission (Kg/m2, m ± SD)	30.7 ± 6.9	29.1 ± 6.2	27.3 ± 5.8	0.003
Comorbidities				
Ischemic etiology of HF % (n)	40 (48.2%)	49 (59%)	38 (45.8%)	0.191
Valvular etiology for HF % (n)	7 (8.4%)	7 (8.4%)	6 (7.2%)	0.947
Atrial fibrillation % (n)	32 (38.6%)	22 (26.5%)	22 (26.5%)	0.150
CABG % (n)	26 (31.3%)	28 (33.7%)	21 (25.3%)	0.475
Stroke % (n)	11 (13.3%)	9 (10.8%)	6 (7.2%)	0.442
Hypertension % (n)	38 (45.8%)	36 (43.4%)	39 (47%)	0.893
Hepatic disease % (n)	9 (10.8%)	7 (8.4%)	6 (7.2%)	0.705
DM on oral medications % (n)	14 (16.9%)	15 (18.1%)	16 (19.3%)	0.922
COPD % (n)	12 (14.5%)	18 (21.7%)	13 (15.7%)	0.418
PVD % (n)	6 (7.2%)	14 (16.9%)	8 (9.6%)	0.123
Mitral regurge % (n)	9 (10.8%)	9 (10.8%)	13 (15.7%)	0.555
Tricuspid regurge % (n)	3 (3.6%)	3 (3.6%)	5 (6%)	0.684
ICD	22 (26.5%)	23 (27.7%)	22 (26.5%)	0.980
Malignancy	3 (3.6%)	5 (6%)	9 (10.8%)	0.171
Vital signs and 6-MWDT on admission				
Supine SBP (mmHg, m ± SD)	107 ± 17	103 ± 16	105 ± 18	0.421
Supine DBP (mmHg, m ± SD)	68 ± 13	65 ± 10	66 ± 12	0.255
Supine heart rate (bpm, m ± SD)	80 ± 15	86 ± 18	83 ± 14	0.084
6-MWD test (feet, m ± SD)	657 ± 319	655 ± 330	632 ± 399	0.965
Laboratory variables on admission				
BNP (pg/mL, m ± SD)	233 ± 82	563 ± 104	1168 ± 248	0.000
BUN (mg/dL, m ± SD)	33.4 ± 22.1	34.1 ± 21.5	37.4 ± 22.2	0.195
Albumin (g/dL, m ± SD)	3.73 ± 0.55	3.6 ± 0.48	3.54 ± 0.44	0.074
Na (meq/L, m ± SD)	136 ± 4.7	137 ± 3.6	136 ± 4.8	0.378
K (meq/L, m ± SD)	4.2 ± 0.6	4.1 ± 0.6	4.3 ± 0.8	0.286
WBC/mcL (m ± SD)	7.8 ± 2.2	9.3 ± 11.7	7.5 ± 2.2	0.447
Troponin I (ng/mL, m ± SD)	0.025 ± 0	0.035 ± 0.02	0.06 ± 0.07	0.209
Echocardiographic data on admission				
EF (%, m ± SD)	21.5 ± 2.1	19.8 ± 8.3	17.7 ± 8.3	0.127
LVEDD (mm, m ± SD)	6.8 ± 1	6.6 ± 1	6.8 ± 1.3	0.439
LVEDV (mL, m ± SD)	262 ± 104	252 ± 93	303 ± 143	0.137
LVESD (mm, m ± SD)	5.9 ± 1.1	5.8 ± 1	6.1 ± 1.3	0.277
LVESV (mL, m ± SD)	212 ± 102	205 ± 83	255 ± 137	0.116
E/A ratio (m ± SD)	2.73 ± 1.44	3 ± 1.27	2.64 ± 1.14	0.474
Deceleration of E velocity (cm/sec, m ± SD)	139 ± 43	132 ± 41	140 ± 76	0.604

BMI: body mass index, CABG: coronary artery bypass graft, DM: Diabetes Mellitus, COPD; chronic obstructive pulmonary disease, PVD: peripheral vascular disease, ICD: implantable cardiac defibrillator, SBP: systolic blood pressure, DBP: diastolic blood pressure, 6-MWD test: 6-minute walk distance test, BNP: B-type natriuretic peptide, EF: ejection fraction, LVEDD: left ventricular end diastolic dimension, LVEDV: left ventricular end diastolic volume, LVESD: left ventricular end systolic dimension, LVESV: left ventricular end systolic volume.

to study the ability of a single BNP measurement to reflect the severity of congestion in patients hospitalized with acute systolic HF after excluding these 2 groups of patients with either normal or extremely elevated BNP, due to the known limited significance of BNP in the later two instances.

2. Methods

This study is a retrospective analysis of a limited access dataset from the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) tria which enrolled 433 patients with acute systolic HF managed with clinical assessment plus pulmonary artery catheterization (PAC) versus clinical assessment alone. All patients had New York Heart Association (NYHA) class IV symptoms and a left ventricular ejection fraction (LVEF) <30% by contrast ventriculography, radionuclide ventriculography or quantitative echocardiography within 1 year before randomization. Results of the ESCAPE trial have been previously published [6]. The Mechanistic Substudy of the ESCAPE trial was designed as Evaluation of Natriuretic Peptides and Troponins as Prognostic Indices and Surrogate Markers for Morbidity and Mortality in Advanced Heart Failure study. This prospective observational study included patients who were part of the ESCAPE trial, and who agreed to provide additional blood samples and thus had more BNP values, than in the original ESCAPE dataset.

The main objective of our study is to determine whether a single BNP value (without longitudinal assessment) can predict the degree of congestion in patients hospitalized with acute systolic HF. Because BNP was more frequently recorded during admission, we performed this analysis using admission BNP. We have shown before that normal or extremely elevated BNP on admission of patients with acute systolic HF has limited ability to predict the degree of congestion, therefore, we have excluded those 2 groups of patients from our analysis. BNP was considered normal if it was <100 pg/mL and extremely elevated if it was >1694 pg/mL (this cutoff represents the 85th percentile of entire cohort BNP level). The remaining cases were divided into tertiles according to baseline BNP value and the severity of congestion was compared across tertiles. The degree of systemic congestion was examined using preset clinical, laboratory and hemodynamic markers of congestion measured by the PAC.