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Original Research Article

Study of Bronchodialatory Effects of B-Type Natriuretic Peptide in Acute Asthma Attacks in Maharashtra Population

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Abstract:

Background: B-type natriuretic peptides (BNP) are independent predictors of cardiac respiratory failure and even death; hence, the effect of BNP in acute asthma attacks was evaluated.

Method: 35 asthma patients were compared with 35 controlled (healthy and volunteers). Asthmatic patients received BNP Via intravenous infusion Two µg/ µg/kg of BNP was injected as a bolus in 60 seconds. Then the infusion of BNP immediately began and was given in 0.01, 0.02, and 0.03 µg/kg/min doses every 30 minutes for the first 1.5 hours. The volunteers in the control group received nebulized salbutamol. Afterwards, peak flow meter findings, hemodynamic parameters, and estimations of the clinical severity of asthma in both groups were taken every 30 minutes.

Results: Respiratory parameters FEV1, PEER, PR per minute, RR per minute, and hemodynamic parameters SBP, DBP, O2, saturation, and dyspnea wheezing had a significant p value ($p<0.001$).

Conclusion: It is concluded that BNP could be a therapeutic option in the treatment of acute asthma attacks, particularly in those with B2 against receptor polymorphism.

Keywords: B-type natriuretic peptide, acute asthma attack, bronchodilator, lifesaving, Maharashtra.

Introduction

Asthma is one of the most common chronic disorders of respiratory tract infection, which increases the frequent hospitalization of patients due to asthma attacks. Many times, these attacks prove to be emergencies and lead to mortality [1]. The natriuretic peptides (NP), B-type natriuretic peptic (BNP), and N-terminal fragment of (NT=pro BNP) are powerful independent predictors of death and adverse events in cardiac failure [2]. It is reported that BNP relaxes tracheal smooth muscle in vitro and is effective in preventing ovalbumin-induced bronchoconstriction in lower animals.

BNP acts as a protective against bronchial hyper responsiveness in asthmatic patients, specifically at the interface between the epithelium and airway smooth muscle cells [3]. Changes from the contractile state to the synthetic state influence the response of smooth muscle cells to drugs and hormones (2). Airway smooth muscle cells derived from asthmatic patients display an altered profile and increased. Production of extracellular matrix proteins, pro-inflammatory mediators, and adhesion receptors, collectively suggesting that airway smooth muscle cells from asthmatic patients have the capacity to actively alter their microenvironment [4]. Hence, an attempt is made to evaluate the molecular and intracellular pathways involved in the response to BNP in acute asthmatic patients.

Material and Method

35 (thirty-five) patients admitted to the emergency department of respiratory medicine of IIIU's IIMSR Medical College hospital warudi, Badnapur (Taluq), Jalna (dist), Maharashtra 431135 were studied.

Inclusive Criteria: The patient's age was between 18-55 years. The patients diagnosed as asthmatics had a history of wheezing, shortness of breath, and coughing. The patients who gave consent for their treatment in writing were selected for study.

Exclusion Criteria: Patients with pulmonary malignancy, LVD (left ventricular dysfunction), esinophilic pneumonia, systemic vasculatures (polyarteritis nodusa), COPD, interstitial lung disease, coronary artery disease, cardiac arrhythmias, and pregnancy, lactation were excluded from the study.

Procedure: 35 asthma patients and 35 controlled (healthy) groups were selected for the study. The peak flow meter and estimation of the clinical severity of asthma were performed in both the control and interventional groups before administering the drug. Then, based on the severity of the asthma attack,

asthma treatment was performed according to standard protocols. Patients with mild to moderate severity of attacks were treated with 2.5 mg of nebulized racemic salbutamol over 20 minutes in three doses as well as 0.5 mg of inhaled ipratropium in doses within 20 minutes. Patients with extremely severe attacks were treated with 5 mg of inhaled racemic salbutamol via inhaler in three doses over 20 minutes, 0.5mg of inhaled ipratropium in three doses over 20 minutes, and 50 mg of oral prednisolone. Patients in the interventional group received BNP via intravenous infusion. For this purpose, 2 µg/kg of BNP was injected as a bolus over 60 seconds. Standard treatment was the base treatment in both groups, but BNP infusion was the additive adjunctive treatment for the case group. BNP infusion consisted of 0.01, 0.02, and 0.03 µg/kg/min each for 30 minutes in the first hours. If the patient had a systolic blood pressure <100 mm Hg on two separate readings, the infusion was discontinued, and the patient was excluded from the study.

Also, if the systolic blood pressure decreased to 20 mmHgµg, the infusion was interrupted. If the patient's systolic blood pressure improved, the infusion was resumed, and the patient was included in the study. Throughout the study, all the patients underwent cardiac monitoring and pulse oximetry. The patient's blood pressure was measured by monitors approximately every 10 minutes. For all patients, drugs were injected through the catheter inserted in the elbow. Patients were treated in a semi-upright position at an angle of 45^{O} degrees. The patients in the control group received standard treatment (nebulized salbutomol). Then peak flow meter findings (peak expiratory flow rates) PEFR and FEV_1, hemodynamic parameters, and the estimation of clinical severity of asthma in both groups were checked every 30 minutes.

The duration of the study was June 2022 to February 2024.

Statistical analysis:

Different parameters of respiration at different intervals were studied in both the controlled and BPN groups and compared with an ANOVA test, and significant results were noted. The statistical analysis was carried out in SPSS software. The ratio of males and females was 2:1.

Observation and Results

Table 1: Comparative study of variables during different periods of time in both the control and -type Nautriuretic groups

- Asthma duration year (mean ± SD) BPN 3.8 (± 1.67) in the BPN group and 3.96 (±2.05) in the control group, and the p value was insignificant ($p > 0.72$).
- FEV1 (mean ±SD) Before Intervention: 1.69 (±0.42) in BPN group, 1.69 (± 0.32) in control, and $p< 0.82$ p value was insignificant.
- At 30^{th} minutes, 60^{th} minutes, 90^{th} minutes, the BPN group and the control group have a significant p value ($p<0.001$).
- PRFR (mean ±SD): Before intervention at 30^{th} minutes, 60^{th} minutes, 90^{th} minutes, the parameters of both the BPN group and the controlled group had a had a significant p value ($p<0.001$).
- PR (per minute): Before intervention at 30^{th} minutes, 60^{th} minutes, 90^{th} minutes, the parameters of the BPN group and the control group had a significant p value ($p<0.001$).
- RR parameters (±SD): Before intervention had an insignificant p value ($p > 0.42$), but 30^{th} minutes, 60^{th} minutes, 90^{th} minutes had significant p values ($p<0.001$).
- SBP (MM µg) (mean ±SD): Parameters between BPN and controlled group before intervention on 30^{th} minutes, 60^{th} minutes, and 90^{th} minutes had significant p value ($p<0.001$).
- DBP (MM µg) (mean ±SD): At 30^{th} minutes, 60^{th} minutes, 90^{th} minutes interval, BPN and the controlled group had a significant p value ($p<0.001$).
- The O2 saturation % study of 0^{th} minutes, 60^{th} minutes, 90^{th} minutes had a significant p value ($p<0.001$).
- Dyspnea: At discharge, speaking sentences had a significant p value ($p<0.001$).
- To phrase 5 (14%), 23 (65.8%), and 7 (20%) in the BPN group and 5 (14%), 26 (74.2%), and 4 (11.4%) in the controlled group
- At discharge, 28 (80%), 7 (20%) in BPN and 28 (80%), 7 (20%) in the controlled group were observed wheezing. Mild 4 (11%), 28 (80%) moderate, and 3 (8.5%) severe were observed only in the PN group.
- At discharge, both groups had the same parameters.

Table 1: Comparative study of variables during different periods of time in both control and B-type Natriuretic group

Variable	BPN	Control	p value
Asthma duration year Mean ±SD	3.8 (± 1.67)	3.96 (±2.05)	P>0.72
FEV1 (mean ± SD) Before intervention	1.69 (± 0.42)	1.69 (± 0.3)	p>0.82
30^{th} min 2.05 (±0.45)	1.98 (± 0.55)	0.54 (±0.1)	P<0.001
60^{th} min 2.37 (±0.42)	2.30 (±0.45)	0.59 (± 0.1)	P<0.001
90^{th} min 2.8 (±0.40)	2.00 (± 0.45)	05 (± 0.1)	P<0.001

PEFR (mean ± SD) Before intervention	290.94 (± 65.9)	280.14 (± 55.05)	P<0.001
30th min 336.1 (±68.2)	1.98 (± 0.55)	0.54 (±0.1)	P<0.001
60th min 378.2 (± 73.5)	336.96 (±52.04)	0.050 (± 0.03)	P<0.001
90th min 429.8 (±77.2)	377.8 (± 52.2)	0.022 (± 0.01)	P<0.001
PR, per min (mean ± SD) Before intervention	90.24 (± 6.02)	92.2 (± 5.14)	P<0.001
30th min 96.6 (±3.05)	96.44 (± 0.44)	0.599 (±0.2)	P<0.001
60th min 99.8 (±4.02)	98.44 (±2.86)	0.499 (± 0.2)	P<0.001
90th min 96.8 (±2.13)	96.46 (± 1.45)	0.599 (± 0.2)	P<0.001
RR per min (±SD) Before intervention	29.63 (± 2.45)	29.86 (± 2.12)	P<0.001
30th min 23 (±1.56)	23.3 (± 1.6)	0.686 (±0.2)	P<0.001
60th min 19.68 (±1.2)	19.36 (±1.6)	0.40 (± 0.3)	P<0.001
90th min 16.2 (±0.77)	16.0 (± 0.76)	0.840 (± 0.2)	P<0.001
SBP mmHG (Mean ±SD) Before intervention	122.24 (± 5.60)	119.4 (± 7.46)	p>0.001
30th min 120.6 (±4.27)	122.6 (± 6.08)	0.552 (±0.2)	P<0.001
60th min 119.76 (±6.86)	120.26 (±4.08)	0.782 (± 0.2)	P<0.001
90th min 119 (±6.55)	119 (± 6.98)	0.872 (± 0.3)	P<0.001
DBP MM/hg (Mean ±SD) Before intervention	79.6 (± 12.56)	79.6 (± 10.8)	P<0.001
30th min 75 (±10.34)	79 (± 4.9)	0.159 (±0.2)	P<0.001
60th min 75.28 (±9.26)	79.6 (±9.2)	0.158 (± 0.3)	P<0.001
90th min 78.26 (±12.98)	80.77 (± 9.70)	0.586 (± 0.2)	P<0.001
O2 Saturation % (mean± SD) Before intervention	90.9 (± 0.77)	91.06 (± 0.89)	P<0.001
30th min 93.4 (±1.15)	93.76 (± 1.18)	0.418 (±0.2)	P<0.001
60th min 95.76 (±0.92)	95.26 (±1.60)	0.238 (± 0.1)	P<0.001
90th min 96.77 (±0.86)	96.9 (± 0.96)	0.858 (± 0.2)	P<0.001
Dyspnea (mean ± SD) Before intervention	4 (± 0.80)	4.26 (± 0.6)	P<0.001
At discharge 0.73 (±0.60)	0.76 (± 0.58)	0.894 (±0.2)	P<0.001
Speaking N (%) Before intervention			
Sentence	5 (14%)	0.668 (0.2%)	P<0.001
Phrase	23 (65.8%)	5 (14%)	P<0.001
	7 (20 %)	26 (74.2%)	P<0.001
At discharge	28 (80%)	28 (80%)	
	7 (20%)	7 (20%)	
Wheezing Before intervention			
Mild	4 (11%)	0	
Inver rate moderate	28 (80%)	3 (85%)	
Severe	3 (85%)	32 (91%)	
At discharge - Mild	31 (9%)	31 (9%)	
Moderate	4 (85%)	4 (85%)	

Discussion

Present study of Bronchodialatory effects peptide in acute asthma patients of Maharashtra. In the comparative study of variables during different periods of time in both control and BPN interventional patients, FEV1, PEFR, PR per minute, RR per minute, SBP, DBP, and O2 saturation dyspnea wheezing at certain intervals had a significant p value ($p<0.001$). These findings are more or less in agreement with previous studies [5,6,7]. Natriuretic peptides (NPS) comprise atrial, brain, and c-type natuiuretic peptides (ANP, BNP, and CNP, respectively), which principally mediate natriuretic, diuretic, vaso-relaxant, and anti-mitogenic responses largely directed at reducing blood pressure and maintaining fluid volume homeostasis [9]. Interestingly, BNP is considered to have a longer plasma half-life and is more resistant to neutral endopeptides. In conditions associated with cardiac hypertrophy and congestive heart failure, both BNP mRNA and circulating BNP levels are markedly increased as compared with ANP and its mRNA levels. Thus, BNP is considered a diagnostic tool that seems to act as an emergency molecule against ventricular overload in disease states. BNP levels are elevated in patients with heart failure, right ventricular failure, pulmonary embolic disease, acute coronary syndrome, valvular heart disease, severe asthma, and COPD [8].

Bronchial muscle relations generally depend on the interaction between action and myosin and the phosphorylation of the myosin light chain. The modulation of the Rho/Rock pathway is intended to mediate a myriad of biological functions and involve contractility. It is reported that BNP-induced relaxation is medicated by epithelial cells and induces early modulation of genes involved in intracellular calcium concentration, leading to smooth muscle con-

traction [10]. After calcium binding, the serine threonine kinase calmodulin directly, phosphorylates and activates myosine activity [11].

It is reported that intravenous nesiritide (BNP) is an effective branchodialator in patients with asthma, and it was revealed that after 180 minutes of nesiritide infusion, FEV1 and FVC expanded to 2.41 and 3.65 L, respectively. It is also noted that the effect of BNP on relaxing bronchial smooth muscle cells is mediated by the epithelium and is associated with rapid changes in EGFR and calcium homeostasis-associated gene levels [12].

Summary and Conclusion

Present study of the Bronchodialatory effects of B-type natriuretic peptide in acute asthma attacks. It is observed and concluded that BNP could be a therapeutic option in the treatment of asthma exacerbations. The present study demands that such clinical trials be conducted on a large number of patients in higher super-specialty respiratory center to combat any adverse reactions to confirm these significant findings and results.

Limitation of study: Owing to the tertiary location of the research center, the small number of patients, and lack of the latest techniques, we have limited findings and results.

This research paper was approved by the ethical committee of JIIU IIMSR Warudi, Badnapur (Tq), Jalna (dist), 431135 Maharashtra.

References

1. Vilkman S, Keisteinen T: Survival and cause of death among elderly chronic obstructive pulmonary disease patients after first admission to the hospital—Respiration 1997, 64; 281-4.
2. Wang TJ, Larson MG: Plasma natriuretic peptide levels and the risk of cardio-vascular events and death, N. Engl. J. Med. 2004, 350, 655–63.
3. Vishwanathan M., Berkman ND: Development of an RTI item bank on the risk of bias and precision of observational studies J. Clin. Epidemoil 2012, 65; 165–78.
4. Beghe E, Verduri A: Echocardiography, spirometry, and systemic acute phase inflammatory propensities in smokers with COPD plus one, 2013, 8e80166.
5. Pandey KN: Biology of natriuretic peptides and their receptors, 2005, 26; 901-32.
6. Ishii K, Murad F: ANP relaxes bovine tracheal smooth muscle and increases CGMP. Am. J. Physol. 1989, 256; 495–500.
7. Zakynthinos E, Kiroulos T: Diagnostic and prognostic impact of brain natriuretic peptide in cardiac and non-cardiac diseases Heart Lung 2008, 37 (4), 275–88.
8. Cargill RI, Lipwarth BJ: Acute effects of ANP and BNP on hypoxic pulmonary vasoconstriction in humans Br. J. Clin. Pharmacol. 1995, 40 (6): 585–90.
9. Cavallazzi R, Nair R, Vasu T: Natriuretic peptides in acute pulmonary embolism: a systemic review, Intensive Care Med. 2008, 34; 2147–2150.
10. Ishii Nomura M: Plasma concentration of brain natriuretic peptide as a biochemical marker for the evaluation of right ventricular overload and mortality in chromic respiratory diseases Clin. Chim. Acta. 2000, 301 (12); 19–30.
11. Leuchte HH, Baumgartner RA: Brain natriuretic peptide is a prognostic in chronic lung disease Am. J. Respir. Crit. Care Med. 2006, 173 (7); 744–50.
12. Matera MG, Calzetta L: Relaxant effect of brain natriureteric peptide in non-sensitized and passively sensitized isolated human bronchi pulm. Pharmacol. Ther. 2009, 22 (6); 478–482.