B-Type Natriuretic Peptide as a Prognostic Indicator of Acute Coronary Syndrome

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ABSTRACT

Introduction: Brain natriuretic peptide (BNP) is release from the cardiac myocytes in response to increased wall stress and volume overload. In this study, we evaluated value of BNP levels in acute coronary syndrome patients.

Material and methods: One hundred and fifty-eight patients with a first chest pain, were examined ECG done and blood samples for BNP were obtained, then echocardiography were done. Linear correlation between two continuous variables was explored using Pearson's correlation (if the data were normally distributed) and Spearman's correlation was used to analyze the value of different baseline characteristics as independent predictors of LV ejection fraction (LVEF).

Results: BNP level in ACS patients were significantly higher (p < 0.001). Regarding to infarct location, the highest BNP level measured in Anteroseptal MI (BNP =1755.71±414.00) pg/ml) and the lowest one indicated in NSTEMI(BNP =387.60 ±259.67 pg/ml (p <0.001).There was significant reverse relation between BNP and EF (p <0.001), (rho = -0.860) and a significant positive relationship between BNP and Troponin I (p = <0.001), (rho = 0.62). Patients with severe systolic dysfunction had the highest BNP levels, while patients with the preserved systolic function had the lowest BNP levelsGroup with EF<30% BNP= 1910.53±362.27 pg/ml, with EF31-40 % BNP=1569.96±555.75 pg/ml, Group with EF 41-50% BNP=637.72±413.62 pg/ml, Group with EF>51% BNP=295.62±159.73 pg/ml, p< 0.001.BNP was the most powerful predictor of mortality in survivors (918.87±731.47) pg/ml vs nonsurvivors (1696.43±465.07) with p<0.001.

Conclusion: After the onset of AMI, BNP blood level can be used as an important predictor for left ventricular dysfunction. Besides echocardiographic calculation, elevation of BNP could be used for quick and easy determination of the left ventricle systolic dysfunction. powerful marker of LV systolic dysfunction and poor prognosis after MI.

Keywords: Brain Natriuretic Peptide, Acute Coronary Syndrome, Left Ventricle Ejection Fraction

INTRODUCTION

First of all de Bold In 1988 discovered BNP in the blood of patients with congestive heart failure.[1] This peptide was named after porcine brain where is first isolated, but after it was realized that heart was its main source.[2]BNP is released from cardiac myocytes due to their stretching, volume overload and high filling pressure. Due to these actions ventricle wall stress occur which initiate to release of BNP precursor i.e. Pre-Pro BNP which cleaves first to Pro-BNP, then to biologically active BNP and inactive aminoterminal fragment, i.e. N-terminal Pro BNP.[3,4]

The most common cause of HF is coronary artery disease, but other well known etiologies are hypertension, valvular disease, cardiomyopathy and myocarditis. For the diagnosis of HF a variety of diagnostic tests is available including assessment of clinical signs and symptoms of HF, laboratory blood tests, radiological examinations, electrocardiography and echocardiography.[5]

Therefore, cardiologists, primary care physicians, and other clinicians became enthusiastic about the role of natriuretic peptides in diagnosis, prognosis and guidance of medical treatment of HF patients.

Release of BNP under influence of integrins, structures at the Z-disc of sarcomeres, that measure stretch of these sarcomeres, after which both peptides will be secreted in equimolar amounts into the circulation.[6]BNP is a 32-amino acid neurohormone synthesized in the form of pre-proBNP, which is firstly cleaved to pro-BNP, and then to active BNP and inactive fragment NT-proBNP. If BNP levelis100-500pg/ml that requires further diagnostic evaluation («grey zone»). If BNP is higher than 500 pg/ml there is high probability of the heart failure.

In general, the suggested normal range for circulating BNP is 0-100pg/ml. These natriuretic peptides may be beneficial in clinical practice since plasma levels of BNP are elevated in patients with HF and are related to the severity of the disease. Circulating BNP acts as an antagonist of the renin angiotensin aldosterone system, and protects the body from plasma overload by inducing diuresis, natriuresis, vascular dilatation and inhibition of the sympathetic nerves system. The half life of BNP is around 20 minutes and the half life of NT-proBNP is around 120 minutes. BNP is known to be cleared from the blood by natriuretic peptide clearance receptors, by neuro endopeptidases and by the kidneys.

In this study we wanted to observe BNP release in acute coronary syndrome and investigate itscorrelation with the left ventricle ejection fraction, localization of infarction, and survival.

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MATERIAL AND METHODS

Study population in this cross-sectional and observational survey included one hundred and fifty-eight patients with acute chest pain. ACS patients (aged54.47±14.63, male =102) who was admitted to ICCU of S.R.N. Hospital Prayagraj, India from January 2019 until June 2020.

These patients were in the initial three days of event. Diagnosis of acute coronary syndrome was confirmed via ST- segment elevation and Non- ST- segment Elevation in initial ECG and cardiac enzymes measurement.

Inclusion criteria-1.Typical ischaemic symptoms and acute coronary syndrome (STEMI, NSTEMI and unstable angina) ECG and elevated levels of cardiac biomarkers (Trop-I) and BNP.2. Patients presentedwith in 24 hours after onset of symptoms.3. Age group > 18 years.

Exclusion criteria- 1.Who were presented after 24 hours of symptoms,2. Age< 18 years, 3. Anemia, 3. Patients with chronic kidney disease,4. Thyrotoxicosis,5. Pulmonary embolism

Demographic information and vital signs, major risk factors (like hyperlipidemia, obesity, diabetes and smoking, hypertension), infarction location and electrocardiogram results were gathered by questionnaire for each patient on admission.

The blood samples for BNP analysis in ACS group were taken within first 24 hours from the beginning of the symptoms. BNP was analyzed using a Triage® BNP test from venous blood collected in EDTA plastic tubes (Ethylenediaminetetraacetic acid). Left ventricle ejection fraction(LVEF) was determined by transthoracic echocardiographic examination using SONOSITE M-TURBO ultrasound operating at 5.0 to 1.0 MHz. The LVEF was calculated from the four-chamber view images.

STATISTICAL ANALYSIS

The statistical analysis was done using SPSS (Statistical Package for Social Sciences) Version 23.0 statistical Analysis Software.The values were represented in Number (%) and Mean±SD. If data were found to be non-normally distributed, appropriate non-parametric tests in the form of Wilcoxon Test were used. Chi-squared test was used for group comparisons for categorical data. In case the expected frequency in the contingency tables was found to be 25% of the cells, Fisher's Exact test was used instead. Linear correlation between two continuous variables was explored using Pearson's correlation (if the data were normally distributed) and Spearman's correlation (for non-normally distributed data). Statistical significance was kept at $p < 0.05$.

RESULTS

Out of 178 patients 158 patients were meet the inclusion criteria of acute coronary syndrome

Demographic data- Demographic data of the current study, such as hypertensive, hyperlipidemic and diabetic subjects, and the other characteristics were comparable (Table 1)

BNP, gender and age- Mean plasma BNP level in male patients (1005.62±734.48)pg/ml, washigher mean plasma BNP level in comparison to female (955.25±768.23) pg/ml($p < 0.673$). In both groups BNP mean rates were not significantly different, according to gender of the participants ($p = 0.673$). There was a weak positive correlation between Age (Years) and BNP, in ACS patients and this correlation was not statistically significant (rho = 0.13, $p = 0.109$). (Table 2)

BNP and MI location- The site of infarction was Anterior Wall in 29.7% (mean BNP level = 1614.11±626.44pg/ml), Inferior Wall15.2% (1320.83±620.98 pg/ml), Anteroseptal wall infarction 4.4% (1755.71±414.00 pg/ml), Lateral Wall3.2%(985.80 ± 737.81 pg/ml), NSTEMI 46.2% (387.60± 259.67) pg/ml of the patients.The highest level of BNP was in Anteroseptal wall infarction MI and the lowest one in NSTEMI. There was a significant difference between the 6 groups in terms of BNP ($X^2 = 92.809$, $p = <0.001$), with the mean BNP being highest in the Type of MI: Anteroseptal group. (Figure 1)

We calculated body mass index (BMI) for ACS and found out significant difference in the number of obese individuals (BMI > 30kg/m2),non-parametric tests (Wilcoxon-Mann-Whitney U Test) were used to make group comparisons. There was a significant difference between the 2 groups in terms of BNP ($W = 726.500$, $p = 0.004$), with the median BNP being highest in the BMI: ≥30 Kg/m2 group. (Table 3)

BNP and Ejection Fraction- The average of left ventricular EF in the patients was 44.59±8.96 %, and there was significant reverse association between BNP level and EF ($p =<0.001$, $r = -0.860$). All patients were divided according to their LVEF in 4 groups: Group I-preserved LV function (EF >51%), Group II-mild LV dysfunction (EF 41-50%), Group III-moderate LV dysfunction (EF 31-40%) and Group IV-severe LV dysfunction (EF <30%).

Patients who have severe left ventricle dysfunction had the highest level of BNP and the patients who have with the preserved systolic function had the lowest levels of BNP. (Table 4)

Figure- 2 showed, the middle horizontal line represents the median BNP, the upper and lower bounds of the box represent the 75th and the 25th centile of BNP respectively, and the upper and lower extent of the whiskers represent the maximum and the minimum BNP in each of the groups.

We also tested possible correlation of BNP and peak troponin value- test of correlation showed statistically significant, There was a strong positive correlation between Troponin I and BNP, and this correlation was statistically significant (rho = 0.62, $p = <0.001$). (Figure 3)

BNP And Outcome

Thepatients those who were expired 8.9%,have more BNP (1696.43 ± 465.07) pg/ml value as compared to survival91.1%, (918.87± 731.47) pg/ml. There was a significant difference between the 2 groups in terms of BNP ($W = 409.000$, $p = <0.001$), with the median BNP being highest in the Outcome: Expired group. (Table 5)

The patients those who were expired have less LVEF (35.00±6.20) % value as compared to survival (45.52±8.64)%. The variable LVEF (%) was not normally distributed in the

2 subgroups of the variable Outcome. Thus, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were used to make group comparisons. There was a significant difference between the 2 groups in terms of LVEF (%) (W = 1651.000, p = <0.001). (table 6)

In myocardial infarction groups the patients those who were expired, have maximum mortality in AWMI (71.4%). There was a significant difference between the various groups in terms of distribution of Type of MI (X^2 = 38.036, p = <0.001). (Table 7)

Variables	Case
Age mean±SD	54.47 ± 14.63.
Gender (%)	
Male	102 (64.6%)
Female	56 (35.4%)
Diabetes (%)	62 (39.2%)
Hypertensive (%)	103 (65.2%)
Smoker (%)	71 (44.9%)
Obese (%)	19 (12.0%)
BNP (pg/mL)	987.77 ± 744.57
Troponin I (ng/mL)	4.25 ± 1.51
BMI (Kg/m2)	24.18 ± 3.65
LVEF (%)	44.59 ± 8.96
LVEF	
≤30%	19 (12.0%)
31-40%	51 (32.3%)
41-50%	40 (25.3%)
>50%	48 (30.4%)

Table-1: Base line data

Parameters	BNP	p value
Age (Years)	Correlation Coefficient (rho) = 0.13	0.109[1]
		0.394[2]
21-30 Years	715.25 ± 703.65	
31-40 Years	928.38 ± 807.76	
41-50 Years	997.69 ± 770.21	
51-60 Years	898.20 ± 662.97	
61-70 Years	1188.06 ± 771.96	
71-80 Years	1075.67 ± 871.94	
81-90 Years	1070.00 ± 464.61	
Gender		0.673[3]
Male	1005.62 ± 734.48	
Female	955.25 ± 768.23	

Table-2: BNP and gender and age

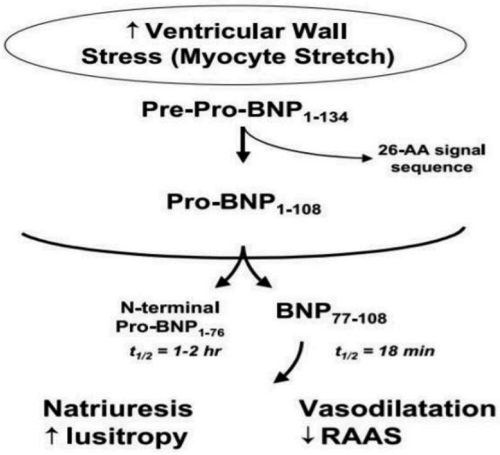

Figure-1: Synthesis and release of B-type natriuretic peptide (BNP). AA - amino acide, RAAS - remmin angiotensin aldosterone system

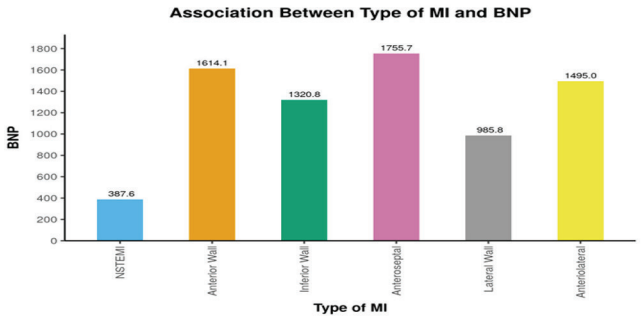

Figure-2: BNP and MI location

BNP	BMI		Wilcoxon-Mann-Whitney U Test	
	<30 Kg/m2	≥30 Kg/m2	W	p value
Mean (SD)	925.36 (739.32)	1473.17 (607.50)	726.500	0.004
Median (IQR)	625.5 (1117.75)	1650 (790)		
Range	102 - 2650	194 - 2300		

Table-3: BNP and adiposity

BNP	LVEF				Kruskal Wallis Test	
	≤30%	31-40%	41-50%	>50%	X^2	p value
Mean (SD)	1910.53 (362.27)	1569.96 (555.75)	637.72 (413.62)	295.62 (159.73)	110.718	<0.001
Median (IQR)	2010 (530)	1600 (875)	529.5 (554.75)	232 (251.25)		
Range	1200 - 2400	369 - 2650	116 - 1900	102 - 647		

Table-4: BNP and different LVEF groups

DISCUSSION

AMI is a major mortality cause and in spite of recent diagnostic and therapeutic improvements; mortality and morbidity rate of this condition is already remained high. Recently, B-type natriuretic peptide (BNP) has been recognized as a useful marker for predicting acute and chronic left ventricular dysfunction. Patients with acute ST- elevation myocardial infarction (STEMI) who had higher levels of BNP have shown to have more mortality outcome.[7]

This study was carried out to determine BNP level in ACS

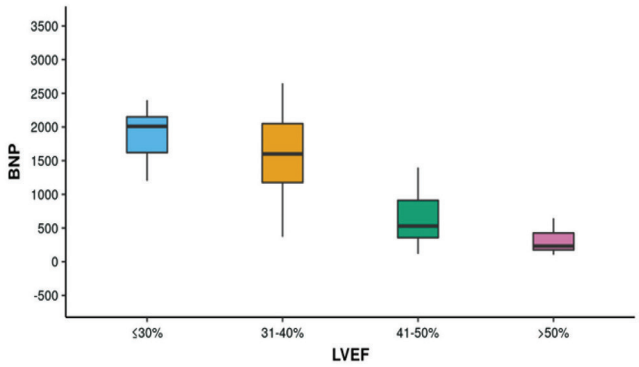

Figure-3: Association of BNP with LVEF

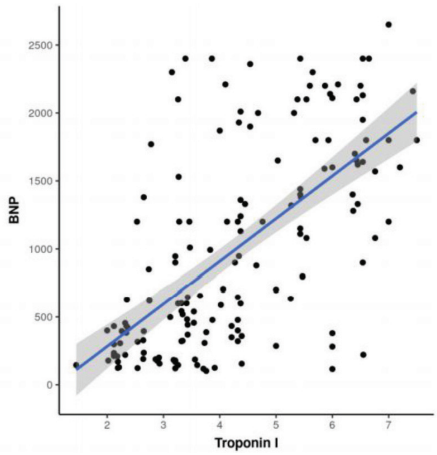

Figure-4: Correlation of BNP and troponin I

BNP	Outcome		Wilcoxon-Mann-Whitney U Test	
	Discharge/Refer	Expired	W	p value
Mean (SD)	918.87 (731.47)	1696.43 (465.07)	409.000	<0.001
Median (IQR)	622 (1232.75)	1580 (802.5)		
Range	102 - 2650	1080 - 2400		

Table-5: BNP and outcome

LVEF (%)	Outcome		Wilcoxon-Mann-Whitney U Test	
	Discharge/Refer	Expired	W	p value
Mean (SD)	45.52 (8.64)	35.00 (6.20)	1651.000	<0.001
Median (IQR)	45 (15)	35 (10)		
Range	30 - 55	25 – 45		

Table-6: BNP and LVEF In Survivor and Non-survivors

Type of MI	Outcome			Fisher's Exact Test	
	Discharge/Refer	Expired	Total	X^2	P Value
NSTEMI	73 (50.7%)	0 (0.0%)	73 (46.2%)	38.036	<0.001
Anterior Wall	37 (25.7%)	10 (71.4%)	47 (29.7%)		
Inferior Wall	23 (16.0%)	1 (7.1%)	24 (15.2%)		
Anteroseptal	6 (4.2%)	1 (7.1%)	7 (4.4%)		
Lateral Wall	5 (3.5%)	0 (0.0%)	5 (3.2%)		
Anteriolateral	0 (0.0%)	2 (14.3%)	2 (1.3%)		
Total	**144 (100.0%)**	**14 (100.0%)**	**158(100.0%)**		

Table-7: Types Of MI In Survivors And Non-survivors

patients, and to evaluate the association of BNP level with left ventricular dysfunction and outcome induced by ACS.

In our study102 (64.6%) of the participants had Gender: Male, 56 (35.4%) of the participants had Gender: Female. Philip Haaf et al in their study reported that median age of the population was 64 years (51- 76) of which 67%(n=717) were male.[8]

The variable BNP was not normally distributed in the 2 subgroups of the variable BMI. Thus, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were used to make group comparisons. The mean (SD) of BNP in the BMI: < 30 Kg/m2 group was (925.36±739.32) pg/ml. The mean (SD) of BNP in the BMI: ≥30 Kg/m2 group was (1473.17±607.50) pg/ml. There was a significant difference between the 2 groups in terms of BNP (W = 726.500, p = 0.004), with the median BNP being highest in the BMI: ≥30 Kg/m2 group.

Recently BNP has been recognized as a useful marker for predicting acute and chronic left ventricular dysfunction. Patients with acute STEMI who have higher levels of BNP have been shown to have worse prognosis.[7]

Morita et al indicated that plasma BNP level was increased in 50 AMI patients in comparison to control group. In the mentioned survey, maximum BNP level was reported, to be prone to rise, in the first 16 hours following admission, more than 50% of normal range, as a second peak.[9]

Yoshida et al reported that female MI patients had significant higher BNP levels, than male MI patients, independent from their age.[10] Comparedwithmales, females showed significantly higher BNPconcentrations (252 vs 155 pg/ml, p < 0.001) despite more preserved left ventricular systolic function (LVEF; 46.0 vs 43.6%, p = 0.03). However, in our study there is slightly higher level of BNP in males as compare to females (1005.62±734.48 vs 955.25±768.23) pg/ml. It seems BNP basal level would be a prognostic LVEF indicator.[11]

In our study subjects, diabetes was found in 62 (39.2%) participants, 103 (65.2%) were hypertensive, 71 (44.9%) of the participants had risk of smoker, 19 (12.0%) were obese. In a study by A.Fazlinezhadet al[12], out of 38 patients, hypertension was found in 17 (39.5%) participants, 8 (18.6%) were diabetics, and 10 (23.3%) was smokers.

In a study Mrinal Kunj et al[13] out of 40 patients 45% of the patients had ALWMI, 15% had ASWMI, 32.5% with IWMI and 7.5% with IWMI ± RWMI. In our study, 73 (46.2%) of the participants had NSTEMI, 47 (29.7%) of the participants had Anterior Wall, 24 (15.2%) had Inferior Wall, 7 (4.4%) participants had Anteroseptal,5 (3.2%) had Lateral Wall and 2 (1.3%) of the participants had Anterolateral.

The mean (SD) of BNP was (987.77±744.57) pg/ml. The median (IQR) of BNP was 702.00 (1293.25). The BNP ranged from 102 - 2650. In a study A. Fazlinezhad et al[12], Mean plasma BNP level in the patient group (3784.57±6344.97) pg/ml was significantly higher than the control group (68.35±69.66 pg/ml) (p< 0.001).

In a study A. Fazlinez had et al, concluded that the site of infarction was anterior in 41.9% (mean BNP level = 4208.92 ± 508.067 pg/ml), inferior-posterior in 25.8% (4436.63 ± 688 pg/ml), isolated inferior wall infarction in 19.4% (598.83 ± 309.867 pg/ml), inferior right ventricle in 12.9% (1462 ± 141.297 pg/ml) of the patients. The highest level of BNP was in posterior-inferior MI and the lowest one in isolated inferior wall MI. There was no significant relationship between BNP levels and location of the infarction (p = 0.13).

In our study, therefore, non-parametric tests (Kruskal Wallis Test) were used to make group comparisons. The mean (SD) of BNP in NSTEMI group was (387.60±259.67) pg/ml, in anterior wall group was (1614.11±626.44) pg/ml, in Inferior Wall group was (1320.83±620.98) pg/ml, Anteroseptal group was (1755.71±414.00) pg/ml, Lateral Wall group was (985.80±737.81) pg/ml, Antero lateral group was (1495.00±134.35) pg/ml.

There was a significant difference between the 6 groups in terms of BNP (X^2 = 92.809, p = <0.001), with the mean BNP being highest in the location of MI: Anteroseptal group and lowest in NSTEMI group. Higher baseline values of BNP were associated with more cardiovascular events.

Our results are consistent with those of Talwar et al.[14]who found BNP elevated to a greater extent in anterior compared to inferior infarction suggesting that a larger area of myocardium was at risk in anterior allocation.

We found statistically significant elevated levels of plasma BNP in acute myocardial infarction. Morita et al.[9] and Richards et al.[15] studied patients of AMI and found a significantly higher BNP plasma levels in comparison with healthy controls.

In our study, we tested possible correlation of BNP and Troponin value. Test of correlation showed statistically significant positive correlation (p<0.001) with medium strength of association-correlation coefficient r= 0.62. Non-parametric tests (Spearman Correlation) were used to explore the correlation between the two variables, as at least one of the variables was not normally distributed. There was a strong positive correlation between Troponin I and BNP, and this correlation was statistically significant (rho = 0.62, p = <0.001). Our study corelated with Azra Durak-Nalbantić et al[16] which have confirmed positive correlation of BNP and peak troponin levels, with medium strength of association (r= 0.441, p< 0.05).

We found that patients with severe systolic dysfunction (LVEF < 30%) had the highest levels of BNP, while group with the normal systolic function (LVEF > 51%) has the lowest level. There was a strong negative correlation between LVEF (%) and BNP, and this correlation was statistically significant (rho = -0.86, p = <0.001). There was a significant difference between the 4 groups in terms of BNP (X^2 = 110.718, p = <0.001), with the median BNP being highest in the LVEF: ≤30% group. Our data is consistent with those of Groenning et al.[17]which showed direct correlation between increase of BNP and decrease of LVEF.

We compared BNP value in survival versus non survival group and found that in nonsurvival group BNP value is higher than in survival group,There was a significant difference between the 2 groups in terms of BNP (W = 409.000, p = <0.001), with the median BNP being highest

in the non survival groups.In our study out of 14 patients in expired groups, anterior wall MI has maximum mortality.

CONCLUSION

The findings of this study confirmed that plasma BNP level could be useful as an important predictor of left ventricular systolic dysfunction, and has as a reverse relation of BNP levels and LVEF is observed in ACS patients. So the measurement of plasma BNP level in early phase of myocardial infarction may be useful as a non-invasive method for identification of individuals, at higher risk of post-MI mortality and also, we can assume LVEF without echocardiography using serum BNP level.

REFERENCES

1. De Bold AJ, Borenstein HB, Veres AT, Sonnenberg H. A rapid and potent natriuretic response to intravenous injection of atrial myocardial extracts in rats. Life Sci 1981;28:89-94.
2. Sudoh T, Kangawa K, Minamino N, Matsuo H. A new natriuretic peptide in porcine brain. Nature 1988;332:78-81.
3. Thygesen K, Mair J, Mueller C, Huber K, Weber M, Plebani M, et al. Recommendations for the use of natriuretic peptides in acute cardiac care, A position statement from the Study Group on Biomarkers in Cardiology of the ESC Working Group on Acute Cardiac Care. Eur Heart J. 2011 doi: 10.1093/eurheartj/ehq509.
4. Sudoh T, Maekawa K, Kojima M, Minamino M, Kangawa N, Matsuo H. Cloning and sequence analysis of cDJNA encoding a precursor for human brain natriuretic peptide in plasma. Clin Chim Acta. 2002;316:129–35.
5. Swedberg K, Cleland J, Dargie H, Drexler H, Follath F, Komajda M, Tavazzi L, Smiseth OA, Gavazzi A, Haverich A, Hoes A, Jaarsma T, Korewicki J, Levy S, Linde C, Lopez-Sendon JL, Nieminen MS, Pierard L, RemmeWJ. Guidelines for the diagnosis and treatment of chronic heart failure: executive summary (update 2005). Eur Heart J 2005;26:1115-40
6. Liang F, Atakilit A, Gardner DG. Integrin dependence of brain natriuretic peptide gene promoter activation by mechanical strain. J Biol Chem 2000;275:20355-60.
7. Mukoyama M, Nakao K, Hosoda K, et al. Brain natriuretic peptide as a novel cardiac hormone in humans. Evidence for an exquisite dual natriuretic peptide system, atrial natriuretic peptide and brain natriuretic peptide. J Clin Invest. 1991;87:1402–12.
8. Philip Haaf, MD, Tobias Reichlin, MD, Nils Corson, MD, Raphael Twerenbold, MD, Miriam Reiter, MD, Stephan Steuer, MD, Stefano Bassetti, MD, Katrin Winkler, MD,, Claudia Stelzig, MSc, Corinna Heinisch, MD, Beatrice Drexler, MD, Heike Freidank, MD, Christian Mueller, MD, FESC. B-type Natriuretic Peptide in the Early Diagnosis and Risk Stratification of Acute Chest Pain. The American Journal of Medicine 2011;124:444-452.
9. Morita E, Yasue H, Yoshimura M, et al. Increased plasma levels of brain natriuretic peptide in patients with acute myocardial infarction. Circulation 1993 ;88:82 – 91S
10. Yoshida A, Kawakami R, Nakanishi M, et al. Gender Difference in B-type Natriuretic Peptide Levels in Patients with Acute Myocardial Infarction, J Card Fail. 2008;14:S158
11. Güneş Y, Okçün B, Kavlak E, et al. Value of brain natriuretic peptide after acute myocardial infarction. Anadolu Kardiyol Derg. 2008;8:182–7
12. A. Fazlinezhad, M. KhademRezaeian, H. Yousefzadeh, K. Ghaffarzadegan and M. Khajedaluee. Plasma Brain Natriuretic Peptide (BNP) as an Indicator of Left Ventricular Function, Early Outcome and Mechanical Complications after Acute Myocardial Infarction. Clinical Medicine Insights: Cardiology 2011:5 77–83.
13. Mrinal Kunj, Bindey Kumar, Anshu Kumar. N-Terminal Pro-Brain Natriuretic Peptide as a Predictor of Complication and Mortality in Acute ST Segment Elevation Myocardial Infarction. Ijcmr 2017;4:1100-1103.
14. S Talwar, I.B Squire, P.F Downie, A.M Mccullough, M.C Campton, J.E Davies, D.B Barnett, L.L Ng. Profile of plasma N-terminal proBNP following acute myocardial infarction. Correlation with left ventricular systolic dysfunction. European Heart Journal, Volume 21, Issue 18, 1 September 2000, Pages 1514–1521.
15. Richards MA, Nicholls MG, Yandle TG, Ikram H, Espiner EA, Turner JG, et al. Neuroendocrine prediction of left ventricular function and heart failure after acute myocardial infarction. Heart. 1999;81:114–120.
16. Azra Durak-Nalbantić, Alen Džubur, Mirza Dilić, Žana Pozderac, Alma Mujanović-Narančić, Mehmed Kulić, Enisa Hodžić, Nerma Resić, Snežana Brdjanović, and Faris Zvizdić. Brain natriuretic peptide release in acute myocardial infarction. Bosn J Basic Med Sci. 2012;12:164–168.
17. Groenning BA, Nilsson JC, Sondergaard L, Kjaer A, Larsson HB, Hildebrant PR. Evaluation of impaired left ventricular ejection fraction and increased dimension by multiple neurohumoral plasma concentrations. Eur J Heart Fail. 2001;3:699–708.

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