International Journal of Innovative Research in Medical Science (IJIRMS)
Volume 08, Issue 09, September 2023,
https://doi.org/10.23958/ijirms/vol08-i09/1741

Original article

Evaluation of Brain Natriuretic Peptide as a Predictor of Contrast Induced Acute Kidney Injury Post Percutaneous Coronary Intervention in Patients with Acute Coronary Syndrome

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Received 07 August 2023; Accepted 25 August 2023; Published 02 September 2023

Abstract

Background: Brain (b-type) natriuretic peptide (BNP) is released into the circulation in response to ventricular dilatation and pressure overload conditions. Studies linked between levels of BNP and short/long term prognosis in patients of acute coronary syndrome (ACS). ***Aim:*** To evaluate the brain natriuretic peptide as a predictor of contrast induced acute kidney injury post percutaneous coronary intervention in patients with acute coronary syndrome. ***Patients and Methods:*** The study included 60 patients who presented to emergency room with diagnosis of ACS syndrome (ST segment elevation myocardial infarction (STEMI), non-ST segment elevation MI (NSTEMI) or unstable angina (UA)) who underwent to percutaneous coronary intervention. All participants subjected to history taking, clinical assessment, ECG, echocardiography and laboratory investigation (serum level of Troponin I, creatinine, blood urea and BNP). ***Results:*** Considering BNP levels at admission, CIN group has significantly elevated BNP level than non-CIN group ($p<0.001$). Value of BNP >69.0 pg/mL can strongly discriminate patients with CIN as AUC was 0.861 and p-value was <0.001 with sensitivity and specificity was81.8% & 92.6% respectively. ***Conclusion:*** BNP with cutoff value >69.0 pg/mL is a simple and easily measurable biomarker that can predict CIN in patients with acute myocardial infarction.

Keywords: ***Acute Coronary Syndrome, Acute Kidney Injury, Brain Natriuretic Peptide, Contrast Induced Acute Kidney Injury***

Introduction

Contrast induced acute Kidney injury (CI-AKI) in cardiac patients who undergo percutaneous coronary intervention (PCI) is common and it results in serious complications which increases the intra hospital morbidity and mortality resulting in increasing the burden over the health system capacity and also the community [1,2].

Detecting the high risk patients to develop post coronary catheterization kidney injury and starting early appropriate prophylactic measures considers a valuable step in controlling the conditions of contrast induced nephropathy (CIN) and hence saving the health organizations resources [3].

Current guidelines provide a number of recommendations to prevent contrast-induced AKI, including the use of iso-osmolar or low-osmolar (water-soluble) contrast media in the lowest possible dose, avoidance of nephrotoxic drugs, and hydration with intravenous (I.V.) crystalloid fluids [4].

Serum creatinine are indicative of renal dysfunction, contrast induced acute kidney injury (AKI) is characterized by a rapid loss of kidney function and increase in serum creatinine of 0.3mg/dl or more than 50% within 48hr after contrast exposure [1,5].

Several risk scores tried to predict conditions of post catheterization contrast induced nephropathy. Mehran risk score is the widely used predictive method has its limitations in cases of non-diabetics, normal kidney and cardiac functions also in conditions when lacking full past medical history such as emergency cases, due to the narrow time window. Hence an objective test to predict the high risk patients for developing post catheterization nephropathy is needed [6].

Brain (b-type) natriuretic peptide (BNP) is a protein synthetized within the ventricular myocardium and released into the circulation in response to ventricular dilatation and pressure overload conditions. Studies linked between levels of BNP and short/long term prognosis in patients of acute coronary syndrome (ACS) [7]. This study evaluated the brain natriuretic peptide as a predictor of contrast induced acute kidney injury post percutaneous coronary intervention in patients with acute coronary syndrome.

Patients and methods

A prospective observational study conducted at Cardiology Department during the period from April 2021 to October 2021.The study included 60 patients who presented toemergency room with diagnosis of ACS syndrome (ST segment elevation myocardial infarction (STEMI), non ST segment elevation MI(NSTEMI) or unstable angina (UA)) who underwent to percutaneous coronary intervention.

Patients with chronic renal disease, heart failure NYHA 3-4, history of severe valvular heart disease, cardiovascular complication as a result of ACS and intra-aortic balloon pump that used before PCI were excluded.

All participants subjected to history taking, clinical assessment, ECG, echocardiography and laboratory investigation

(serum level of Troponin I, creatinine, blood urea and BNP). We analyzed BNP using a commercial BNP ELISA Kit (Biomedica, Vienna, Austria).Venous blood was obtained by direct venipuncture and collected into plastic tubes-Lavender top containing potassium ethylene diamine tetra-acetic acid (EDTA). In amounts that ranged from 1 to 3 ml. All samples were centrifuged and frozen within 1-7 hours from sampling and thawed only once at the time of analysis. Plasma was tested for BNP using the immunoenzymatic assay, a point-of-care device that uses an immunoassay technique a commercial BNP ELISA Kit (Biomedica, Vienna, Austria).

All data analyzed using statistical package for social sciences (SPSS) version 22 (SPSS Inc, Chicago, USA). For qualitative data, frequency and percent distributions was calculated. For quantitative data, mean, standard deviation (Sd) was calculated. significance was defined as P value < 0.05. The following tests were used; Student T test, Mann-Whitney U test and Chi- Square test.

Results

This study involved 60 patients with ACS, their age ranged from 46 to 77 years with mean age ±SD being 60.48± 7.80 years. The majority of cases were males (60 %). The studied patients were divided according to development of contrast-induced nephropathy (CIN) which is 50% rise in serum creatinine from baseline or an increase of 0.3 mg/dL within 48-72 hours following intravenous contrast administration [8] into two groups; group (A) involved 33 cases who developed contrast-induced nephropathy and group (B) involved 27 cases who did not develop contrast-induced nephropathy. Age was significantly higher in CIN group compared to group B (p= 0.006). Patients in CIN group were also significantly more frequently had diabetes mellitus than group B (p= 0.025). NYHA II was significantly higher in CIN group (p= 0.02) (**Table 1**).

Regarding LVEF and number of vessel lesions, patients in CIN group were more likely to have multiple stents, with interventions of multiple lesions in >1 vessel (p<0.001). There was no statistically significant difference between the two groups regarding LVEF (p>0.05) (**Table 2**).

Considering BNP levels at admission, CIN group has significantly elevated BNP level than non-CIN group (p<0.001) (**Table 3**).

Regarding logistic regression analysis for factors predicting of contrast-Induced nephropathy, there was significant association between age (p= 0.035) and serum BNP at admission (p= 0.015) with occurrence of CIN. However, there was no significant association between hypertension, DM, LVEF, creatinine and BUN at admission with occurrence of CIN (p>0.05) (**Table 4**).

According to the validity for BNP >69.0pg/mL in predicting contrast induced AKI post primary percutaneous coronary intervention in patients with ACS, BNP can strongly discriminate patients with CIN as AUC was 0.861 and p-value was <0.001 with sensitivity and specificity was81.8% & 92.6% respectively (**Figure 1**).

Table 1: Comparison between the two studied groups regarding demographic and clinical data.

		CIN group (No. = 33)		Non-CIN group (No. = 27)		Test value	p-Value
		No.	%	No.	%		
Age (years)	**Mean± SD**	63.18± 6.60		57.19± 8.0		Z_{MWU}=2.76	**0.006****
	Median	64.0		56.0			
	Range	49.0 - 77.0		46.0 - 70.0			
Gender	**Male**	16	48.5%	20	74.1%	X^2= 3.06	0.08
	Female	17	51.5%	7	25.9%		
Diagnosis	**STEMI**	14	42.5%	7	25.9%	X^2= 1.126	0.289
	NSTEMI	19	57.5%	18	66.6%	X^2= 0.206	0.650
	UA	0	0%	2	7.04%	X^2= 0.752	0.386
Comorbidities	**Hypertension**	21	63.6%	16	59.3%	X^2= 0.006	0.936
	DM	12	36.4%	3	11.1%	X^2= 5.05	**0.025***
	CKD	0	0.0%	0	0.0%	-	-
NYHA classification	**NYHAI**	27	81.8%	27	100.0%	X^2= 5.46	**0.020***
	NYHA II	6	18.2%	0	0.0%		
SBP (mm/Hg)	**Mean± SD**	116.80± 7.48		116.0± 6.45		Z_{MWU}=0.278	0.781
	Range	110.0 - 155.0		100.0 - 130.0			
DBP (mm/Hg)	**Mean± SD**	74.80± 5.10		74.40± 5.83		Z_{MWU}=0.144	0.885
	Range	70.0 - 105.0		55.0 - 80.0			

**p≤0.05 is considered statistically significant, **p≤0.01 is considered high statistically significant, SD= standard deviation, X2: Chi- Square test and ZMWU: Mann-Whitney U test*

Table 2: Comparison between the two studied groups regarding LVEF and number of vessel lesions.

		CIN group (No. = 33)		Non-CIN group (No. = 27)		Test value	P-value
		No.	%	No.	%		
LVEF (%)	**Mean± SD**	57.24± 8.46		60.56± 7.65		Z=1.53 MWU	0.126
	Median	54.0		61.0			
	Range	45.0 - 73.0		49.0 - 73.0			
Number of vessel lesions	**One**	5	15.2%	25	92.6%	X^2= 32.59	**<0.001****
	Two	10	30.3%	2	7.4%	X^2= 3.540	0.060
	Three	13	39.4%	0	0.0%	X^2= 11.36	**0.001****
	Four	5	15.2%	0	0.0%	X^2= 36.43	0.100

**p≤0.05 is considered statistically significant, **p≤0.01 is considered high statistically significant, SD= standard deviation, X2: Chi- Square test and ZMWU: Mann-Whitney U test*

Table 3: Comparison between the two studied groups regarding admission BNP

		CIN group (No. = 33)		Non-CIN group (No. = 27)		Test value	P-value
		No.	%	No.	%		
BNP (pg/mL)	**Mean± SD**	136.09± 102.16		52.33± 73.66			**<0.001****
	Median	102.0		39.0		Z MWU=4.79	
	Range	21.0 - 392.0		13.0 - 406.0			

**p≤0.05 is considered statistically significant, **p≤0.01 is considered high statistically significant, SD= standard deviation, ZMWU: Mann-Whitney U test*

Table 4: Independent predictors of contrast-induced nephropathy

Parameters	B	S.E.	Wald	P-value	Odds ratio (OR)
Age	.248	.117	4.455	**0.035**	1.281
HTN	-41.709-	4107.873	0.000	0.992	0.000
DM	-42.404-	4107.874	0.000	0.992	0.000
LVEF	2.499	8.489	0.087	0.768	12.169
Adm. Creatinine	1.322	2.740	.233	0.629	3.752
Adm. BUN	.098	.082	1.413	0.235	1.103
Adm. BNP	.066	.027	5.934	**0.015**	1.069

B: Regression coefficient; S.E.: Standard error, CI: Confidence interval
HTN: Hypertension, DM: Diabetes mellitus; LVEF: Left ventricular ejection fraction; BUN: Blood urea nitrogen; BNP: Brain natriuretic peptide

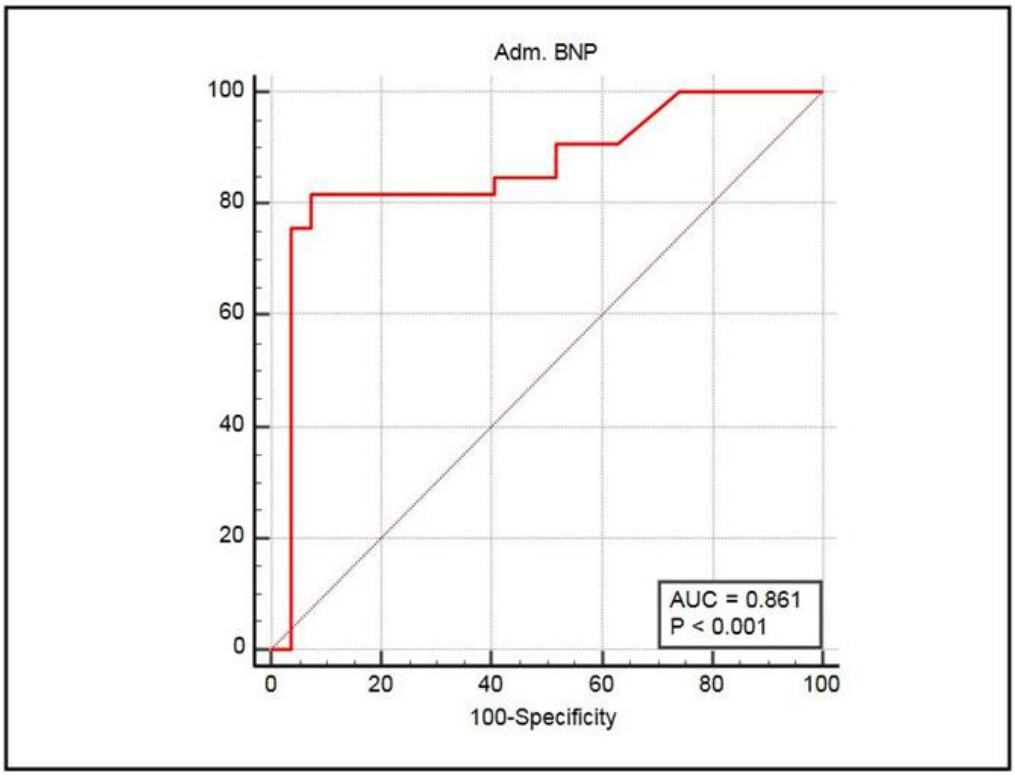

Figure 1: ROC curve for BNP in predicting contrast induced acute kidney injury post primary percutaneous coronary intervention in patients with acute coronary syndrome

Discussion

Acute coronary syndrome (ACS), including unstable angina (UA), non-ST elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI), generally results from atherosclerotic plaque rupture or superficial plaque erosion [9]. Despite great progress in the treatment of ACS over the past few decades, ACS is still a major cause of death worldwide [10].

Regarding laboratory results at admission of the studied cases we found that the mean level of serum creatinine was 0.77± 0.23 mg/dl and the mean BUN was 29.63± 9.12 mg/dl. The mean BNP level was 98.40± 99.08 pg/ml. BNP levels at admission were markedly elevated in patients who developed CIN compared to patients who did not develop CIN.

In agreement with our results the study by Agarwal et al.[11] reported that the mean level of serum creatinine was 1.02±0.3 mg/dl and the mean urea was 26.01±10.3 mg/dl. The mean NT-proBNP level was 2620.46±4073.3 pg/ml. There was statistically significant difference between studied groups regarding mean serum creatinine (p<0.001), while there was statistically significant difference regarding urea.

Our results were supported by the study by Jarai et al.[12] reported that the mean level of serum creatinine was 1 mg/dl, so they found that CI-AKI developed in 131 patients (13%). BNP levels at baseline were markedly elevated in patients who developed versus did not develop CI-AKI (median versus 62.6 pg/mL, respectively; P<0.0001).

Furthermore, our results agree with the study by Naruse et al.[13] revealed that the mean creatinine-based estimated glomerular

filtration rate was 0.91 (0.83–0.99) and the mean NT-proBNP was 1120 (230–4024) pg/mL, there was statistically significant difference between studied groups regarding mean creatinine and NT-proBNP (p<0.001).

Logistic regression analysis for factors predicting of contrast-induced nephropathy. There was statistically significant association between age (p= 0.035) and serum BNP at admission (p= 0.015) with occurrence of contrast-induced nephropathy. There was no statistically significant association between hypertension, DM, LVEF, creatinine and BUN at admission with occurrence of contrast-induced nephropathy (p>0.05).

The study by Jarai et al.[12] support our results as they reported that there were statistically significant association between contrast-induced acute kidney injury and age, BNP, LVEF, and hemoglobin while no statistically significant association was found regarding creatinine. In disagreement with our results, they found no significant interactions between examined subgroups and the association between baseline BNP and the development of CI-AKI.

In contrast to our results the study by Agarwal et al.[11] reported that even though diabetes has been described as a risk factor for CIAKI, they did not find a significant association between diabetes or hypertension and CI- AKI in their study.

We finally presented the validity for BNP in predicting contrast induced acute kidney injury post percutaneous coronary intervention in patients with acute coronary syndrome, and found that BNP >69.0pg/mL can strongly discriminate patients with contrast-Induced nephropathy as AUC was 0.861 and p-value was <0.001 with sensitivity and specificity was81.8% & 92.6% respectively.

And the study by Akgul et al.[14] reported that a BNP value of 88.7 was identified as an effective cut-off point for 6-month all-cause mortality [area under curve =0.82, 95% confidence interval (CI) 0.73–0.92, p<0.001] and AKI (area under curve =0.65, 95% CI 0.57–0.73, p<0.001). A BNP of >88.7 pg/ ml yielded a sensitivity of 77.8% and a specificity of 77.6% for 6- month all-cause.

While the study by Goussot et al.[15] concluded that ROC analysis showed that the threshold of Nt-proBNP for the prediction of CIN was 204 pg/mL, yielding sensitivity at 80% and specificity at 59%. These findings strongly suggest the specific interest of this biomarker for CIN risk in younger patients.

Furthermore, the study by Kurtul et al.[16] revealed that NT-proBNP ≥2149 pg/mL measured on admission had a 79.4% sensitivity and 74.3% specificity in predicting CIN.

Our study limited by small sample size and follow up by renal functions was not possible, hopefully future studies can be able to assess long term evaluation of the kidney function.

Conclusion

BNP with cutoff value>69.0pg/mL is a simple and easily measurable biomarker that can be used in risk stratification of a poor prognosis and the development of CIN in patients with acute myocardial infarction. These patients with high BNP levels can be monitored closely to determine the in hospital adverse outcomes and mid-term prognoses.

Conflict of interest

None

Funding

The study was funded by the authors.

Data availability

Data would be available upon reasonable request.

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