ORIGINAL ARTICLE

SIGNIFICANCE OF LEFT VENTRICULAR END DIASTOLIC PRESSURE FOR RISK STRATIFICATION OF CONTRAST-INDUCED ACUTE KIDNEY INJURY AFTER PRIMARY PERCUTANEOUS CORONARY INTERVENTION

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Objectives: The objective of this study was to determine the significance of left ventricular end diastolic pressure (LVEDP) for risk stratification of contrast-induced acute kidney injury after primary percutaneous coronary intervention (PCI).

Methodology: This cross-sectional study was conducted at the largest cardiac care center of the Pakistan. Consecutive patients presented to the emergency department and diagnosed with ST-segment elevation myocardial infarction (STEMI) undergone primary PCI were included. Serum creatinine level were obtained at baseline and after 48 to 72 hours and contrast induced nephropathy (CIN) was recorded. Receiver operating characteristic (ROC) curve analysis was performed to assess the predictive strength of LVEDP and area under the curve (AUC) was reported.

Results: Out of 488 cases, seventy-two (14.8%) patients developed CIN. Left ventricular end diastolic pressure predicted CIN with an AUC of 0.582 [95% CI: 0.510 to 0.654], the optimal cut-off value of LVEDP ≥ 20 mmHg yielded overall classification accuracy of 49.2% (95% CI: 44.7% to 53.7%) with sensitivity of 66.7% (95% CI: 54.6% to 77.3%) and specificity of 46.1% (95% CI: 41.3% to 51.1%). The predictive accuracy increased as patients ejection fraction decreased, LVEDP predicted CIN with an AUC of 0.623 [95% CI: 0.540 to 0.707] among patients with LVEF ≤40%, while, the AUC of LVEDP for predicting CIN was 0.504 [95% CI: 0386 to 0.622] for patients with LVEF > 40%.

Conclusion: Elevated intra-procedural LVEDP (≥20mmHg) is independently associated with an increased risk of CI-AKI for patients undergoing cardiac catheterization and PCI, especially in the setting of reduced LVEF (≤40%).

Keywords: ST-segment elevation myocardial infarction, primary percutaneous coronary intervention, left ventricular end diastolic, contrast induced nephropathy

Citation: Ammar A, Khowaja S, Kumar R, Kumar V, Hussain A, Ahmed S, Siddiqui MN, Rasool M, Sial JA, Saghir T. Significance of Left Ventricular End Diastolic Pressure for Risk Stratification of Contrast-Induced Acute Kidney Injury after Primary Percutaneous Coronary Intervention. Pak Heart J. 2022;55(03):247-252. DOI: https://doi.org/10.47144/phj.v55i3.2135

INTRODUCTION

Ischemic heart disease (IHD) is the predominant cause of morbidity and mortality across the globe. Despite decline in mortality in developed countries, mortality still remains high in third world countries.[1] Ischemic heart disease can have grave consequences on our health. It is associated with systolic and diastolic dysfunction. Diastolic dysfunction can result in inefficient emptying of left atrium, inadequate filling of left ventricle, diminished capacity of heart to maximize cardiac output while exercising, heightened pulmonary artery pressure, and fluid overload. The importance of systolic dysfunction on coronary artery disease (CAD) is indispensable; it enhances the vulnerability to adverse major cardiovascular events.[2] Similarly, the left ventricle dysfunction (LVD) results in secondary development of high pulmonary arterial pressure, which in turn worsens the outcome of myocardial infarction (MI).[3]

Earlier studies have shown that left ventricular end diastolic pressure (LVEDP) truly represents pulmonary arterial wedge pressure (PAWP) in absence of pulmonary arterial disease.[4] Hence, this can be easily deduced that PAWP could similarly highlight the LVEDP to evaluate blood volume, reveal cardiac physiology and observe the intravenous fluid administration while hydrating. Essentially, PAWP is a best bedside indicator of hemodynamics. For example, PAWP is regarded as an ideal predictor for managing intravenous fluid volume for prevention of pulmonary edema. As such, to avert any adverse major

cardiovascular events, it is imperative to identify the prognostic value of LVEDP and early cognizance of factors that could influence LVEDP, which will help us in earliest detection the prognosis.[5] Although non-invasive methods like Doppler echocardiogram have been embraced to guage LVEDP, catheterization is still considered as the gold standard.

Contrast-induced nephropathy (CIN) is quite common and one of the most frequent complications of post percutaneous coronary intervention (PCI). Adequate hydration has been considered as an important approach to prevent CIN, and adequate intravascular volume is the only accepted preventive measure.[6] The common potential risk factors that can culminate in CI-AKI are old age, female gender, anemia, hypotension, diabetes mellitus (DM), congestive heart failure (CHF), chronic kidney disease (CKD), high volume contrast and emergency procedures. Reducing the exposure to contrast is the ideal way to lessen the possibility of CI-AKI. However, intravenous volume loading or oral hydration remains first line when administering high volume contrast is inevitable.[7,8] Of late, findings published in A Maastricht Contrast-induced Nephropathy Guideline (AMACING) trial demonstrated that suppressing preemptive intravenous hydration with normal saline was not any lesser to administering fluids; there was no significant difference in incidence of CI-AKI.[8]

The objective of this study was to determine the significance of left ventricular end diastolic pressure for risk stratification of contrast-induced acute kidney injury after primary percutaneous coronary intervention (PCI).

METHODOLOGY

This cross-sectional study was conducted at the largest cardiac care center of the Pakistan. Study was approved by the ethical review committee of the institution and due to observational nature of the study verbal consent was obtained from all the patients. Required number of consecutive patients presented to the emergency department and diagnosed with ST-segment elevation myocardial infarction (STEMI) between January 2020 and December 2020 were included. Inclusion criteria for the study were either gender, age above 17 years, presented in emergency department within 12 hours of symptom onset, diagnosed with STEMI, and undergone primary PCI. Patients who refused primary PCI were excluded from the study. STEMI was diagnosed as per the fourth universal definition of myocardial infarction.

Data for the study was collected with the help of a structured Performa consisted of demographic details, risk factor, clinical characteristics, angiographic, and procedural characteristics. Patient's clinical history was obtained regarding hypertension, smoking status, diabetes mellitus, and family history of IHD, stroke, chronic kidney disease, congestive heart failure, obesity, and prior PCI. At presentation data regarding duration of symptom, killip class, and hemodynamic parameters (heart rate and blood pressure) were obtained. All the primary PCI procedures were performed as per the current practice guidelines and institutional protocols by consultant cardiologists with at least two years of working experience. Serum creatinine (mg/dl) level were obtained at baseline and after 48 to 72 hours and criteria for CIN was either absolute difference of 0.5 mg/dL or higher or more than 25% increase in serum creatinine (mg/dL) level at 48 to 72 hours as compared to baseline. Data regarding angiographic findings (disease burden, thrombus burden, culprit artery, and localization of disease), procedural characteristics (contrast volume, fluoroscopy time, use of IABP and stent) and hemodynamic parameters (LVEDP and left ventricular ejection fraction (LVEF)) were obtained.

Data was entered and analyzed using IBM SPSS version 21, patients were stratified by the development of CIN and demographic and clinical characteristics were compared for patients with and without CIN. Continuous response variables were represented by mean ± standard deviation (SD) and compared by applying independent sample t-test. Categorical response variables were represented by frequency (%) and Chi-square test was performed to assess the associations. Receiver operating characteristic (ROC) curve analysis was performed to assess the predictive strength of LVEDP for risk stratification of development of CIN and are under the cure (AUC) was calculated. Considering the influence of LVEF on the accuracy of LVEDP, stratification was made at 40% cutoff for LVEF and ROC analysis results were obtained. A p-value ≤ 0.05 was taken as statistical criteria for significance.

RESULTS

A total of 488 primary PCI cases were included in this analysis, 393 (80.5%) patients were male and mean (AGE) of the study sample was 56.75 ± 11.45 years with 28.3% (138) elderly (≥ 65 years) patients. A total of seventy-two (14.8%) patients developed contrast induced nephropathy (CIN).

Patients who developed CIN were older (61.64 ± 10.69 vs. 55.91 ± 11.38; p<0.001), and more likely to present in cardiac arrest (11.1% vs. 4.8%; p=0.034) and

require intubation (27.8% vs. 14.4%; p=0.005) as compared to patients without CIN. They were more likely to have history of stroke (5.6% vs. 1%; p=0.005), chronic kidney disease (5.6% vs. 1.4%; p=0.023), and congestive heart failure (2.8% vs. 0.5%; p=0.046). Baseline blood glucose level (199.13 ± 99.11 mg/dL vs. 172.76 ± 76.71 mg/dL; p=0.010) and serum creatinine (1.24 ± 0.48 mg/dL vs. 1.07 ± 0.41mg/dL; p= 0.003) were significantly higher for CIN patients, while, baseline mean hemoglobin (12.7 ± 2.14 mg/dL vs. 13.87 ± 2.01 mg/dL; p<0.001) was low among CIN patients as compared to non-CIN patients.

High thrombus burden (grade ≥4) was more common among patients with CIN (84.7% vs. 69.7%; p=0.009), also incidence of placement of temporary pacemaker (16.7% vs. 8.2%; p=0.023) was higher among patients who developed CIN as compared to those who didn't. The mean fluoroscopic time was significantly higher in CIN patients (18.23 ± 11.09 minutes vs. 15.59 ± 8.86 minutes; p=0.025). Similarly, mean LVEDP (21.88 ± 7.36 mmHg vs. 20.04 ± 6.8 mmHg; p=0.037) was significantly higher for CIN groups. Demographics and clinical characteristics of patients stratified by development of CIN are presented in Table 1.

Table 1: Demographics and clinical characteristics of patients stratified by development of contrast induced nephropathy

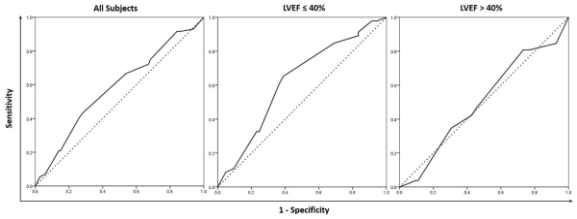
Characteristics	Total	Contrast Induced Nephropathy (CIN)		P-value
		No CIN	CIN	
Total (N)	**488**	**416 (85.2%)**	**72 (14.8%)**	**-**
Baseline demographic				
Male	80.5% (393)	81.3% (338)	76.4% (55)	0.336
Mean age (years)	56.75 ± 11.45	55.91 ± 11.38	61.64 ± 10.69	<0.001*
Old (≥ 65 years)	28.3% (138)	25% (104)	47.2% (34)	<0.001*
Presentation				
Symptom to ER arrival time (minutes)	298.5 ± 190.6	291.9 ± 184.8	336.4 ± 218.8	0.067
Door to balloon time (minutes)	102.4 ± 65	101.5 ± 61.4	107.4 ± 83.2	0.479
Total ischemic time (minutes)	400.9 ± 216.6	393.4 ± 209	443.8 ± 253.2	0.068
Systolic blood pressure (mmHg)	124.6 ± 24.2	125.3 ± 23.4	120.6 ± 28.5	0.122
Heart Rate (bpm)	85.1 ± 21.8	85.1 ± 21.4	84.8 ± 23.8	0.709
Killip Class III or IV	16.2% (79)	14.9% (62)	23.6% (17)	0.064
Cardiac Arrest	5.7% (28)	4.8% (20)	11.1% (8)	0.034*
Intubated	16.4% (80)	14.4% (60)	27.8% (20)	0.005*
History				
Hypertension	52% (254)	50.5% (210)	61.1% (44)	0.096
Smoking	25.4% (124)	26.4% (110)	19.4% (14)	0.208
Diabetes mellitus	39.5% (193)	38% (158)	48.6% (35)	0.089
Family history of IHD	2.7% (13)	2.9% (12)	1.4% (1)	0.467
Stroke	1.6% (8)	1% (4)	5.6% (4)	0.005*
Chronic kidney disease	2% (10)	1.4% (6)	5.6% (4)	0.023*
Congestive heart failure	0.8% (4)	0.5% (2)	2.8% (2)	0.046*
Obesity	0.8% (4)	0.5% (2)	2.8% (2)	0.046*
Prior history of PCI	8% (39)	7.7% (32)	9.7% (7)	0.558
Clinical characteristics				
Radial access	63.1% (308)	65.9% (274)	47.2% (34)	0.002*
Multi-vessel disease	69.5% (339)	68.3% (284)	76.4% (55)	0.167
Culprit – LAD	52.9% (258)	54.3% (226)	44.4% (32)	0.286
Culprit – RCA	33.6% (164)	33.2% (138)	36.1% (26)	
Culprit – LCx	12.1% (59)	11.1% (46)	18.1% (13)	
Culprit – LM	1.4% (7)	1.4% (6)	1.4% (1)	
High thrombus burden (≥4)	71.9% (351)	69.7% (290)	84.7% (61)	0.009*
TPM implanted	9.4% (46)	8.2% (34)	16.7% (12)	0.023*
IABP used	7.2% (35)	6+.7% (28)	9.7% (7)	0.364
Stent used	91.2% (445)	91.3% (380)	90.3% (65)	0.768
Fluoroscopic time (minutes)	15.98 ± 9.26	15.59 ± 8.86	18.23 ± 11.09	0.025*
Contrast volume (ml)	120.84 ± 42.83	119.42 ± 42.33	129.03 ± 45.1	0.079
Hemodynamic parameters				
LVEDP (mmHg)	20.31 ± 6.91	20.04 ± 6.8	21.88 ± 7.36	0.037*

LVEDP < 20 mmHg	44.3% (216)	46.2% (192)	33.3% (24)	0.043*
LVEDP ≥ 20 mmHg	55.7% (272)	53.8% (224)	66.7% (48)	
LVEF (%)	38.22 ± 9.43	38.32 ± 9.42	37.64 ± 9.53	0.574
LVEF ≤ 40%	61.1% (298)	60.6% (252)	63.9% (46)	0.595
LVEF > 40%	38.9% (190)	39.4% (164)	36.1% (26)	
Laboratory assessments				
Arrival - blood glucose (mg/dL)	176.65 ± 80.83	172.76 ± 76.71	199.13 ± 99.11	0.010*
Arrival - hemoglobin (mg/dL)	13.7 ± 2.07	13.87 ± 2.01	12.7 ± 2.14	<0.001*
Arrival - creatinine (mg/dL)	1.1 ± 0.43	1.07 ± 0.41	1.24 ± 0.48	0.003*
72 Hours - creatinine (mg/dL)	1.29 ± 0.77	1.09 ± 0.39	2.39 ± 1.29	<0.001*
Clinical outcome				
In-hospital mortality	3.7% (18)	2.9% (12)	8.3% (6)	0.024*

*ER=emergency room, IHD=ischemic heart diseases, PCI=percutaneous coronary intervention, LAD=left anterior descending artery, RCA=right coronary artery, LCx=left circumflex, LM=left main, TPM=temporary pacemaker, IABP=intra-aortic balloon pump, LVEDP=left ventricular end diastolic pressure, LVEF=left ventricular ejection fraction, *significant at 5%*

In the overall study sample, LVEDP predicted CIN with an AUC of 0.582 [95% CI: 0.510 to 0.654], the optimal cut-off value of LVEDP ≥ 20 mmHg yielded overall classification accuracy of 49.2% (95% CI: 44.7% to 53.7%) with sensitivity of 66.7% (95% CI: 54.6% to 77.3%) and specificity of 46.1% (95% CI: 41.3% to 51.1%). As evident from Figure 1, the predictive accuracy increased as patients ejection fraction decreased, LVEDP predicted CIN with an AUC of 0.623 [95% CI: 0.540 to 0.707] among patients with LVEF of less than or equal to 40%, while, the AUC of LVEDP for predicting CIN was 0.504 [95% CI: 0386 to 0.622] for patients with LVEF of more than 40%.

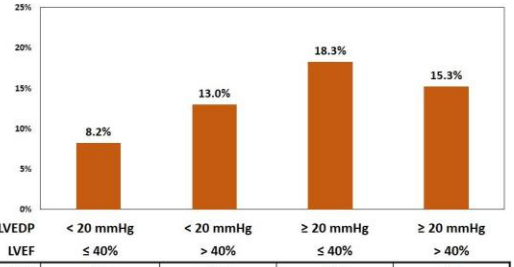
Figure 1: Receiver operating characteristics (ROC) analysis for LVEDP to predict development of contrast induced nephropathy (CIN), stratified by LVEF



LVEDP=left ventricular end diastolic pressure, LVEF=left ventricular ejection fraction

Impact of the interaction between LVEF and LVEDP in predicting CIN is presented in Figure 2. The incidence of CIN in patients with ≤ 40% LVEF increased from 8.2% to 18.3% at LVEDP of < 20 mmHg and ≥ 20 mmHg respectively, while, this increment with respect to incremental LVEDP was marginal (13.0% to 15.3%) for patients with LVEF of more than 40%.

Figure 2: Impact of LVEDP and LVEF on incidence rate of CIN



Total	85	131	213	59
CIN	7	17	39	9
Incidence rate	8.2%	13.0%	18.3%	15.3%

LVEDP=left ventricular end diastolic pressure, LVEF=left ventricular ejection fraction

DISCUSSION

This study aimed to evaluate the role of LVEDP in predicting the contrast induced AKI (CI-AKI) in individuals undergoing primary PCI. With AUC of 0.582, we found LVEDP to have moderate predictive value for CI-AKI with the optimal cutoff point of ≥20 mmHg, sensitivity of 66.7% and specificity of 46.1%. Interestingly, the predictive value of LVEDP was comparatively higher for patients with LVEF of ≤40% (AUC 0.623) in contrast to >40% (AUC 0.504). Furthermore, at LVEDP of <20mmHg and ≥20mmHg, the CIN incidence increased from 8.2% to 18.3%, respectively, in patients with reduced LVEF. This relationship was found to be marginal; from 13.0% to 15.3%, for patients with preserved LVEF. This finding was in agreement with that of reported by Liu C et al.[9]; a similar positive relationship between LVEDP and incidence of CI-AKI and influence of LVEF was communicated. In the present study, LVEDP of ≥20mmHg was witnessed to be an independent predictor of CI-AKI (AUC 0.64). The association was in fact even stronger for patients with reduced ejection

fraction (odds ratio of 4.08). Earlier, inverse relationship between CI-AKI and LVEDP has also been cited by Gu and colleagues.[10]

Research literature suggests a significant relationship between CIN and vulnerability to adverse outcome for patients with acute myocardial infarction (AMI), irrespective of LVEF, with normal kidney function.[11-13] Prompt identification of CIN is essential and therefore several tools have been proposed to predict CIN. Certain demographic and clinical characteristics of patients like old age, female gender, baseline deteriorated kidney function, anemia, diabetes mellitus (DM), congestive heart failure (CHF), and AMI at presentation have been reported to be associated with increased propensity to develop CI-AKI post percutaneous interventions.[13]

In a study conducted by Abe et al., they found a reverse relationship between LVEDP and occurrence of CI-AKI in individuals undergoing primary PCI.[13] The study subjects received an intravenous normal saline infusion 4 hours prior to PCI which continued for 24 hours post PCI. Afterwards, loop diuretic (20mg intravenous furosemide) was administered. The study did not mention fluid volume status including urine output. It is clinically relevant in furosemide-naive patients because an intravenous loop diuretic might significantly increase the urine output and will result in substantial reduction of volume, which might heightened the risk of developing CI-AKI, particularly in patients with normal or low baseline LVEDP. In addition, primary PCI patients with CHF were omitted by Gu et al. Such patients frequently possess potential factors to develop CI-AKI and high LVEDP.[10]

Of late, a randomized clinical trial (ATTEMPT) evaluated the impact of aggressive hydration with intravenous infusion of normal saline in comparison with general hydration guided by LVEDP on CIN and key clinical outcomes in post-STEMI patients.[14] The main aim of the ATTEMPT trial was to offer valuable evidence to direct the optimal hydration strategy for patients with STEMI undergoing primary PCI. In present cohort, high risk patients recognized by traditional risk factors were given low volume contrast, highlighting physicians' efforts to prevent incidence of CI-AKI. Nevertheless, individuals with high LVEDP were provided with the similar volume of contrast in comparison with their counterparts, which should not be the case. This suggests the lack of knowledge regarding elevated LVEDP as an important risk factor of CI-AKI. Hence, it is imperative to elevate the cognizance of the relationship between high LVEDP (≥20mmHg) and high propensity to develop CI-AKI in individuals undergoing primary PCI. In fact, LVEDP should be considered for potential inclusion for future research projects of clinical CI-AKI risk evaluation models.[15-17]

This was an observational study conducted at a single center in a limited number of patients. Although the current study indicated that LVEDP was a fine parameter to evaluate risk of CIN, its wide application has been limited because of the invasive nature of measurement procedures. Due to time limitation and resources availability the LVEDP was taken with MP catheter which is acceptable but not gold standard method.

CONCLUSION

Elevated intra-procedural LVEDP (≥20mmHg) is independently associated with an increased risk of CI-AKI for patients undergoing cardiac catheterization and PCI, especially in the setting of reduced LVEF (≤40%).

AUTHORS' CONTRIBUTION

AA, SK, RK, VK, AH, SA, MNS, and MR: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. JAS, and TS: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

Conflict of interest: Authors declared no conflict of interest.

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